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COMPRISING TEN VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY

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VOLUME X.

NERVOUS AND MENTAL DISEASES

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TABLE OF CONTENTS.

NERVOUS DISEASES.

PAGE

- The Neurone Theory 5
- Symptomatology 6
- Reflexes 11
- Neuroses 19
  - Neurasthenia 19
  - Hysteria 23
  - Epilepsy 30
  - Migraine 35
  - Sydenham's Chorea 37
- Diseases of the Brain and Meninges 41
  - Cerebrospinal Meningitis 41
  - Cortical Localization 64
  - Aphasia 73
  - Arteriosclerosis 79
  - Senility 80
  - Cerebral Birth Palsies 83
  - Apoplexy and Hemiplegia 84
  - Encephalitis and Sinus Thrombosis 93
  - Cerebral Abscess 94
  - Cerebral Tumor 98
  - Optic Thalamus Syndrome 119
  - Cerebellum 120
  - Medulla Oblongata 134
- Diseases of the Spinal Cord 135
  - Trauma 136
  - Tabes Dorsalis 138
  - Acute Ascending Myelitis 141
  - Acute Poliomyelitis 143
  - Landry's Paralysis 148
  - Amyotrophic Lateral Sclerosis 154

PAGE
Friedreich's Hereditary Ataxia 154
Multiple Sclerosis 156
Syringomyelia 160
Tumors 164
Diseases of the Peripheral Nerves 169
Cranial Nerves 169
Spinal Nerves 179
Multiple Neuritis 191
Miscellaneous Nervous Diseases 196

MENTAL DISEASES, 209

General Paralysis 219
Paranoia 224
Dementia Precox 227

NERVOUS DISEASES.

THE NEURONE THEORY.

Nerve Degeneration and Regeneration. During the past year attention has been diverted from the neurone theory to an investigation of nerve-elements. However, W. D. Halliburton,[1] in the Oliver-Sharpey Lectures, discusses the processes of nerve-degeneration and regeneration, and presents besides a critical analysis of the present controversy in regard to autogenetic nerve-regeneration, a carefully recorded set of experiments, performed by Mott, Edmunds, and himself. These may be classed under five headings:

1. Experiments in which union of central and peripheral ends was prevented, such as enclosing the proximal end of each popliteal nerve in caps made of small sterilized drainage tubes.
2. Experiments on transplanted pieces of nerves.
3. Experiments on degeneration of regenerated fibers.
4. Experiments on the rate of medullation in regenerating nerves.
5. Experiments on the influence of stimulus on regeneration.

These experimental methods thus approach the subject in diversified ways, and in no one case was evidence forthcoming of autoregeneration. "The facts recorded, taken in conjunction with those published by such observers as Cajal and Langley and Anderson, form, on the other hand, strong pieces of evidence in favor of the Wallerian doctrine that new nerve fibers are growths from the central ends of divided nerve trunks. The experimental facts recorded by those who, like Bethe and Kennedy, hold the opposite view are susceptible of easy explanation, mainly on the lines emphasized by Langley and Anderson of acci-

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(1) Lancet, May 4 and 11, 1907; British Med. Jour., same dates.

dental and unnoticed connection of the peripheral segments with the central nervous system by means of other nerves cut through in the operation. If such connection is effectually prevented, real regeneration of structure and restoration of function never occurs. Moreover, the regenerated fibers always degenerate in a peripheral direction, and in a peripheral direction only, when the link that binds them to the central nervous system is again severed."

In an appended note Halliburton states that Bethe[1] now admits that "in adult animals no regeneration of medullated fibers occurs autogenetically; he does not, however, state at what age animals lose their power of autogenetic regeneration, nor explain how it is that after a certain date an animal repairs a divided nerve in the exactly contrary manner to that it would have adopted if the nerve had been cut before that date. He also again admits that a regenerated nerve when cut, degenerates in a peripheral direction only, but denies that degeneration has anything to do with direction of growth."

SYMPTOMATOLOGY.

Pallesthesia. R. T. Williamson,[2] discussing the vibrating sensation in nervous diseases, reports twelve illustrative cases. In early tabes dorsalis the vibrating sensation was lost on the legs (at the malleoli, inner surface of the tibia, and soles of the feet), when sensations of touch, pain, and temperature were distinctly felt. In some of these cases there was no ataxia, in others it was present. Similar results were obtained in early suspected tabes, when other symptoms were very few. The author's conclusions follow:

"1. The vibrating sensation is a delicate test for detecting slight impairment of sensation. The vibrating sensation may be lost when other forms of sensation (to tactile impressions, pain, and temperature) are felt quite well, or are only very slightly impaired. This is sometimes the case in early tabes, in slight peripheral neuritis, and often in diabetes mellitus.

"2. In diabetes mellitus the vibrating sensation may be

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(1) Pflüger's Arch., 1907, cxvi, p. 385.
(2) British Med. Jour., July 20, 1907.

lost on the feet, or feet and legs, when there are no other nervous symptoms; but often the latter are present.

3. In diseases strictly limited to the motor structures, the vibrating sensation is not lost even at an advanced period of the disease.

4. In cases of paraplegia from spinal caries, and occasionally in spinal syphilis, the loss of the vibrating feeling may be the only objective symptom of affection of sensation, at an early stage of the disease.

5. In hemianesthesia, if the vibrating feeling is lost when the foot of the tuning-fork is placed on the edge of the sternum on the side of the tactile anesthesia, but felt on the other side, the case is one of hysterical or functional hemianesthesia, or of malingering; while in hemianesthesia due to organic disease the vibrating sensation is felt when the foot of the vibrating tuning-fork is placed on the edge of the sternum on the side of the tactile anesthesia.

Quinquaud's Sign. L. Minor[1] has made a very exhaustive study of Quinquaud's sign, and its frequency among abstainers, alcoholics, hysterics, tabetics, and victims of various nervous diseases. He tells an interesting story of the origin of the sign. One day Quinquaud asked his students to spread their fingers and place the tips at right angles against the palm of his hand. They did so, and without saying a word, he looked at two of them ironically, and shook his head warningly at some others. A month later he died, without explaining his act. Maridort, one of the students, set out to discover the essential feature of the test, and its meaning. To the sign he gave the name of his master, and declared that it prevailed among alcoholics. The sign is a feeling experienced by the investigator as though the ends of the metacarpal bones and phalanges bumped on one another in a series of tiny impacts. Minor has improved on the technic of observation, making use of the phonendoscope, and a resonator of soft wood, 9 by 12 by 3 cm. Thus he distinguishes six degrees of intensity of the sign. His conclusions follow, somewhat condensed:

1. Quinquaud's sign is neither a specific nor pathog-

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(1) Berlin. klin. Woch., May 6, 13, 20, 27, 1907.

nomonic sign of alcoholism, and is frequently present in marked degree in moderate drinkers and total abstainers.

2. Among nervous maladies it is rare in exophthalmic goiter, paralysis agitans, arthritis deformans, and in certain stages of early hemiplegia. It is absent in old hemiplegia with contractures (doubtless also in muscular contractures of other origin), in Dupuytren's contracture, and in musculospiral paralysis. It is more frequent in tabes dorsalis than in any other disease; hysteria comes next, and alcoholism ranks third or even fourth.

3. At present Quinquaud's sign cannot be regarded as physiologic, despite the frequency of its occurrence. Much favors that view, however, and it may be regarded as a very acute sign of varying hypotonus of muscles.

4. The phenomenon of flickering in the interosseous muscles described by Lauschner, was found in tabes dorsalis. Still the question as to what muscles evoke the sign must at present remain unanswered, because it has been noted in high degrees of atrophy of the interossei. This fact indicates the possibility of participation of the long muscles of the forearm.

5. There is no proof that the sign originates in either the tendon-sheaths or in the joints. The tuning-fork experiment might indicate that the phenomenon is one of very coarse vibrations, or that there are molecular vibrations transmitted from the bones of the patient to the hands of the investigator, or to the resonator.

6. The best *tactile* method of making the test is to have the patient spread his fingers, and at the same time extend them, then place the tips of the second, third, and fourth fingers on the observer's palm at an angle of 45 degrees. The best *acoustic* method is to use a resonator in the form of a closed box of soft wood, placed on a firm table, to have the patient apply his finger-tips to it as to the palm, and to listen for the tiny shocks of the bumping phalanges with a phonendoscope. This method is more convenient, exact, uniform, and productive of results, than the tactile one.

Ataxia. Ataxia of a new type is described by M. Allen Starr[1] as occurring in a patient of 66 years of age, asso-

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(1) Med. Record, May 18, 1907.

ciated with nerve deafness doubtless the result of progressive atrophy of the auditory nerve. "In walking, his feet are placed irregularly, the steps being of uneven length and the body swaying without any special tendency to fall in any one direction. His gait is that of cerebellar disease rather than of locomotor ataxia, as there is none of the stamping of the feet or throwing of the legs in irregular directions. When seated there is absolutely no indication of any incoördination either in the movement of the legs or of the arms. His writing is perfectly good, and there is no trace of ataxia excepting in the act of standing and walking. It is impossible for him to stand with his eyes closed, but while this lack of balance is very marked, it is not attended by any vertigo, and it appears to be the only symptom that is present.

"It seems probable that along with atrophy of the cochlear division of the auditory nerve, which has produced his deafness, there has been a progressive atrophy of the vestibular division of the auditory nerve, cutting off from his brain those impulses from the labyrinth which are necessary for the preservation of balance."

Cytodiagnosis. F. P. Rous[1] has made a careful study of cerebrospinal fluid, as to its pressure, protein-content, and the number and character of its cells, both from normal individuals and from those suffering from nervous diseases. Only four factors are of value: (1) The subarachnoid pressure; (2) the protein-content; (3) the cell content, and (4) the differentiation of the cells found.

1. Subarachnoid pressure was measured by joining a vertical tube directly to the puncture needle, and estimating the pressure in terms of the cerebrospinal fluid itself. The tube used was thick-walled, of approximately 1.5 mm. in diameter. The capillary error is 20 mm. of normal cerebrospinal fluid, and to measure a pressure of 500 mm. requires only 0.9 c.c. of the fluid. Pulse and respiratory waves in the column show its direct pressure connection with the subarachnoid space. When these are lacking, an error in technic must be looked for.

2. The protein-content was determined by a modification of the Esbach method. A tube similar to the Esbach

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(1) Am. Jour. Med. Sciences, April, 1907.

tube, but with a base of only 9 mm. so that but 5 or 6 c.c. of cerebrospinal fluid would be required for the test, was adopted, and from it readings were taken as from an Esbach tube. Only the clear supernatant fluid after centrifugalizing should be used.

3. The cell-content was determined by a specially devised method: "A saturated aqueous solution of methyl violet (5B) is drawn up in the tube of a 1 to 100 *mélangeur* until it has filled four of the decimal divisions on the tube, and then it is followed with the fresh cerebrospinal fluid until the bulb of the instrument is full. This furnishes a mixture, slightly darker than ordinary "Toison solution," containing 996 parts in 1000 of the fluid for examination. In this way the leucocytes are stained violet, the erythrocytes preserve their hue, and with the usual blood-counting chamber the cells can be rapidly enumerated."

4. The cell character is determined by centrifugalizing a portion of the cerebrospinal fluid, decanting, spreading, drying, staining the coagulum, and making a differential count.

The cerebrospinal fluid was examined in the following cases: (1) Cases of disease other than nervous, (2) cases of nervous disease without abnormality of the fluid, (3) cases of tuberculous meningitis, (4) cases of purulent meningitis due to various infections, and (5) cases without purulent or tuberculous meningitis, but with abnormality of the cerebrospinal fluid. Practically, the fluids obtained in groups one and two were identical in character. In these two combined groups 20 observations were made. The pressure varied from 70 to 300 mm., with an average of 210 mm. a figure much above Krönig's normal range between 120 and 180 mm. All observations were taken with the patient lying on his side, the knees drawn up, and the head on a level with the vertebral column. The salient conclusion is that cerebrospinal pressure varies within wide limits. The protein-content varied but little; the lowest amount was 1-6 gram per liter, the highest $\frac{3}{8}$ gram, the average of 1-3 gram. The number of cells varied from 0.5 to 6.5 per c. mm., with an average of 2.75. In no specimen were cells entirely absent. The differential counts showed mononuclears of all sizes, but no polymorphonuclear cells were observed except in contamination with blood.

In group three seventeen punctures were made in nine cases of tuberculous meningitis. Pressure at the initial puncture varied between 240 and 550 mm. of the fluid itself, with 365 mm. as an average. Later punctures usually showed a lower pressure, once 50 mm., so that the extremes of pressure were 50 and 550 mm. The mean was 308 mm. The protein-content was increased, ranging from ½ gram per liter to over 10 grams. The high figure was obtained in a case showing postmortem an extraordinary gelatinous meningitis. In the other eight cases the protein did not exceed 1¾ grams per liter and averaged slightly less than 1 gram. The cell content was much increased. Never below 43 per cubic mm., the count ran as high as 686 with an average of 213; and in all cases and at all punctures but one there were over 100 cells per cubic mm. The differential counts brought out little beyond the general tendency to lymphocytosis. Cell percentages were the same in the first fluid and in the last obtained at the same puncture.

Most importance attaches to the cerebrospinal fluid as a whole in tuberculous meningitis. Protein, pressure, and cell-content are all largely increased, even in specimens which to the naked eye are quite limpid. The cells found in the differential count are lymphocytes. "While a diagnosis of tuberculous meningitis *intra vitam* cannot be absolute without discovery of the characteristic bacilli or a positive result on animal inoculation, yet a clear or slightly turbid cerebrospinal fluid under increased tension, with over 100 cells per cubic mm., among which lymphocytes predominate, and an increase in protein on Esbach's test, strongly point to the lesion. A long-standing purulent meningitis in which the many polymorphonuclear cells have been replaced by a few mononuclears, and an occasional case of paresis, are the only other conditions likely to yield such a fluid. The power to confirm by these means, a diagnosis of tuberculous meningitis is not so important as is that to rule it out absolutely. * * * Mere failure to find tubercle bacilli in the cerebrospinal fluid does not enable one to dismiss with certainty the possibility of tuberculous meningitis, nor does the finding that cells in the fluid are few by the method of centrifugalization and an estimation of cell number to the oil-immersion field. But it would seem that one can affirm the absence of the disease when the fluid conforms to the normal as regards pressure, protein, and cell-content, as here determined."

Group four consisted of cases of "purulent meningitis" due to *Streptococcus pyogenes, Diplococcus pneumoniæ,* and *Diplococcus intracellularis.* Twelve punctures were made in six cases. Immediately on puncture the cerebrospinal fluid was obviously abnormal; indeed, from some specimens, a coagulum separated so rapidly as to prevent a cell count. During the acute stage, pressure, protein, and cell-content are all high—protein and cell-content higher usually than in tuberculous meningitis. Among the cells present, polymorphonuclears predominate. Pressure in the acute cases ranged from 290 to 560 mm., averaging 390 mm.; protein ranged from 1 gm. to 5 gm. and averaged 2.5 gm. per liter; and the cells varied from 1300 per cubic millimeter, or 20,000 and 30,000, to such numbers as make up a thick pus. With chronicity came mononuclears and a fluid that could not be distinguished by protein, pressure, cell number and character from that of tuberculous meningitis. It is questionable whether a distinction from tuberculous meningitis can be made, when a process, later to become purulent, is slowly gaining headway, or when a purulent lesion is focal in character.

Group five consisted of cases without purulent or tuberculous meningitis, but with abnormalities in the cerebrospinal fluid. These cases were miscellaneous, consisting of three instances of brain tumor, two of uremia, one of convulsions in association with otitis media, two of syphilitic insanity, one from syphilitic hemiplegia, one from syphilitic chronic spinal meningitis, two from hydrocephalic children, and three other cases, one being undiagnosticated. So variable were the findings that each set of cases is discussed by itself, and a proper summary is at best too vague to be worth while. More important is the fact that Rous was able to demonstrate that the so-called "degenerated cells," or "cellular transformation" reported by various authors are truly degenerated cells. This he was able to do by incubating cerebrospinal fluids for varying lengths of time, and comparing slides with controls made before incubation. The author draws the following conclusions:

1. The methods described herewith offer the means for a much more careful clinical study of the non-purulent, cerebrospinal fluid than has heretofore been usual. Determinations should always be made of the pressure in the subarachnoid cavity, of the protein-content of the fluid, the number of cells per c.mm., and their kind.

2. By these means a specimen may often be pronounced pathologic which under the ordinary examination appears normal. Admixture of blood does not necessarily render a fluid valueless.

3. Normal fluids differ but little in quantitative cell-content. It is doubtful whether cells are ever wholly lacking, and up to 7 per c.mm. may provisionally be set as the range in specimens not pathologic. The normal protein-content, too, lies within well-defined limits. On the other hand, pressure variations which depend on factors other than a lesion of the central nervous system are wide.

4. Except along the broadest lines there is nothing specific in the permutations of protein, pressure, and cells. But along these lines the fluid from cases of tuberculous meningitis is characteristic, as compared with that from cases of paresis or of cerebrospinal syphilis, and these also differ from the normal fluid. If a case be suspected to be one of tuberculous meningitis, but the fluid prove to be normal as regards pressure, protein, and cells, the disease may be ruled out.

5. The "clear elements," "cellular degenerations," "pseudoendothelial cells," often noted, are definitely proved to be degenerated cells.

REFLEXES.

The Cerebral Element. G. L. Walton and W. E. Paul[1] discuss the cerebral element in reflexes and its relation to the spinal element. They affirm that "both the brain and the cord must play a part in the production of the deep reflexes. The higher arcs tend to produce a more subdued and deliberate reflex, while the lower arcs tend to produce an active, instantaneous and violent reflex. In

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(1) Jour. Nerv. and Ment. Diseases, November, 1906.

health the resultant is a comparatively moderate reflex, varying in different individuals as the cerebral or the spinal type predominates. In organic disease the partial withdrawal of the cerebral influence causes the spinal type of reflex, namely the exaggerated reflex. The spinal arc is, however, incapable of alone sustaining the function as shown by the fact that complete withdrawal of cerebral influence causes abolition of all reflexes.

"It is now generally agreed that sudden destruction of the upper cord abolishes the reflex. There is still some difference of opinion on the question whether upon the gradual, complete withdrawal of cerebral influences reflexes may not be preserved as claimed by Raymond and Cestan.

"The control of the superficial reflexes varies somewhat from that of the deep reflexes. The disappearance of the superficial reflexes after the cerebral lesion indicates cerebral control; the fact that they do not become exaggerated in disease of the pyramidal tract shows that they have little if any representation in the spinal cord. The control of the Babinski reflex more nearly approximates that of the deep than that of the superficial reflexes."

Seventeen illustrative cases are adduced. The authors conclude as follows: "The deep reflex is a resultant of the activity of cerebral and spinal arcs, the longer arcs tending to produce a deliberate and moderate reflex, the shorter arcs an active and violent reflex. The deep reflex varies in healthy individuals and in the neuropsychoses according to the predominant influence of the longer or shorter arcs.

"In disease of organic origin the partial withdrawal of the higher influence causes the spinal type of the deep reflex, but the complete withdrawal of the higher influence causes abolition of the deep reflexes, since the spinal arc alone is incapable (in man) of sustaining the burden. On re-establishment of the higher influence the reflexes return, the spinal type predominating if the re-establishment is partial, the normal type if it is complete.

"Initial lessening or loss of deep reflex in the paralyzed parts is the rule in apoplexy. This condition persists for a period varying from half an hour to a number of days, after which these reflexes become normal or assume the spinal (exaggerated) type according as the return of cerebral influence has been complete or partial. In the exceptional cases of apoplexy with initial exaggeration of deep reflexes the withdrawal of cerebral influence has been from the first incomplete."

Paradoxic Reflex of Gordon. Alfred Gordon[1] reports a second case giving anatomic proof of the value of the paradoxic reflex which goes by his name. The first case was presented by F. X. Dercum,[2] and by means of the reflex a diagnosis of localized hemorrhagic pachymeningitis was aided. Dercum observed the presence of the reflex on the side opposite the lesion before the operation, its disappearance after removal of the clot, and its reappearance when a second hemorrhage occurred, the latter being verified at the necropsy. In this case the Babinski phenomenon was not distinct.

This second case of Gordon's was that of a young man of 23 years of age, with a cicatrix on the right parietal region, showing pronounced mental hebetude and somnolence. The left knee-jerk was exaggerated, and the paradoxic reflex was obtained with the greatest facility. Babinski's toe-sign, Oppenheim's reflex, and ankle-clonus were all absent. An operation by Da Costa restored the patient to health, and in six weeks the paradoxic reflex, looked for by Gordon, was found wanting, as was also the increased knee-jerk. Gordon believes that his early conclusion is warranted, and it is here repeated: "The paradoxic reflex is a sign of value in the beginning of an organic affection of the motor system or in cases of irritation. While the Babinski sign is of inestimable value in cases with a definitely established lesion of the motor system, the paradoxic reflex, to say the least, is a sign of transient irritation or of a slight lesion of the same motor pathway."

The Integrative Action of the Nervous System. A summary of the year's progress in neurology is incomplete without mention of Charles S. Sherrington's book (London, 1906), an excellent review of which appears in the *Edinburgh Medical Journal,*[3] written by W. G. Smith. In

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(1) Amer. Jour. Med. Sciences, August, 1907.
(2) Jour. Nerv. and Mental Dis., September, 1906.
(3) May, 1907.

the introductory chapter nervous integration is defined. The unit mechanism in integration is the reflex arc, and the unit integrative act is the reflex. First the functions of the simple reflex are determined, and subsequently those of the complex one. The author then takes up the characteristics given to the simple reflex first by the receptor, and next by the conductor. The modification attributable to the receptor is selective excitability, the threshold being lowered to certain stimuli. The functions of the conductor are next considered. It is pointed out that part of the reflex arc lies in gray matter, and the time difference between conduction in nerve-trunk and gray matter is due to the presence of a surface of separation between the nerve-cells in the synapse. In regard to reflex latency, the most significant fact is the relatively great lessening in the reflex under the operation of weaker stimuli. The after discharge is shown to be heightened by prolongation and by intensification of the stimulus. Irreversibility of direction in the reflex arc, the difference between the rhythm of excitation and response, and the refractory phase in the reflex arc, are all attributed to the synaptic junction. In opposition to other writers, Sherrington finds that the intensity of reflex action is dependent in some degree on the strength of stimulus. The increase of end-effect with the increase of the stimulus is due to a graded activity of a unified center.

Inhibition is regarded as an essential feature in the process of excitation. When an animal can act in many ways, harmonious action is possible only when one definite action prevails, and the opposing reflexes are suppressed; this is reciprocal innervation, and the inhibition and relaxation are coördinate reciprocal factors in one united response. The site of inhibition is not the motor neurone, but is some internuncial mechanism. In dealing with the compounding and interaction of reflexes the dominating principle is the "common pathway." The afferent neurone is a private path; the internuncial and the efferent final paths, are common pathways. If common paths exist there is no qualitative differences in nerve impulses. Wherever found the common path is a mechanism for coördination; by its means there is put at the disposal of each receptor an extensive system of effectors; and on the other hand, one effector may be employed by many receptors.

The first point to be noted regarding *simultaneous* combinations of reflexes is irradiation; with increase of stimulus there may be a far-reaching spread of reflexes. The character of the spread seems dependent on the nature of the synapse. As to *successive* combinations it is shown that through the operation of "spread," each reflex favors the appearance of the next allied reflex. Thus the maintenance of a series of harmonious reflexes is assured when the stimuli are approximately similar. But if the stimuli are sufficiently dissimilar there is interference between the first reflex and that which is called forth by the second stimulus. Such interference is specially marked in alternating and compensatory reflexes; in the former there is a sequence of antagonistic reflexes possessing the common pathway in turn, and in the latter a return of equilibrium. Of the factors contributing to determine which one of competing reflexes will prevail, the most potent is intensity, but there are to be noted also "spread" or immediate induction, successive induction, fatigue, and the specific character of the stimulus. Successive induction is in many ways the reverse of "spread"; it prepares the way, and reinforces not an allied but an antagonistic reflex. It is exemplified in ankle-clonus. By the action of successive induction, alternating reflexes tend to be maintained even apart from definite stimulation.

What is termed fatigue is, in many respects, functional in nature, a process adapted to prevent a particular line of activity, a particular effector from being continuously and exclusively engaged by any one receptor. Thus fatigue secures serial variety of reaction. Finally, it holds an important position among reflexes connected with receptors stimulated by agencies threatening damage—the noci-ceptive receptors; their hold on animal activity is prepotent and imperative, approached by only one other reflex, the sexual one.

Reflexes as reactions of adaptation are then considered. The conception of purpose in reflex action is useless in physiology so long as it is external; it becomes important, however, when it enables an animal to dominate its environment. An account of pseudo-affective reflexes next follows, in which purpose seems clearly indicated, and an account of experimental work which renders it highly probable that visceral and motor sensations are not the primary factor, but are rather a secondary and reinforcing factor in the development of emotion.

After reference to the fact that the motor cortex is entirely precentral, Sherrington points out that the reactions here initiated are very similar to those of the spinal cord. Strikingly contrasted with such spinal and cortical reactions is the muscular activity displayed in persistent "decerebrate rigidity," which ensues when the hemispheres are cut off form the brain stem. Evidently there is a mechanism somewhere between hemispheres and medulla which controls the musculature in a manner apart from that by which it is actuated by cranial and spinal reflexes. This mechanism produces a persistent tonus ordinarily, which is seen in an exaggerated form in decerebrate rigidity. Thus there are two systems of motor innervation; the one gives steady tonic attitude or posture, the other gives transient phasic movements, which, whenever they appear, must inhibit this tonic contraction. The real difference between cortical and spinal reactions appears to be in the receptors. The cerebrum is intimately and exclusively related to receptors which mediate reaction to distant objects—distance receptors. It is these receptors and the actions initiated by them which control the behavior of the animal as a whole, and through them the cerebrum has attained the control of the whole organism.

Following this analysis of reflexes is a statement of the physiologic position and dominance of the brain. First there is a rapid review of the reactions of the unicellular organism, the multicellular organism with diffuse nerve-net, and the organism showing a synaptic system. By the synaptic system and the common paths which it provides, there is secured the unification of the primary series of segments comprising the organism, and coördination of action. It is plain that the receptors in the head segments will tend to develop, because their development will be of preëminent advantage to the organism; in giving the animal indications regarding objects at a distance they acquire the psychic character of projicience. While the other receptors are related to the action of the after-segments, these distance receptors relate the whole individual to its environment. Thus they give rise to precurrent reactions, while the spinal receptors initiate consummatory reactions. By means of these projicient receptors the range of experience is immensely increased for the head segments. Just as the cerebrum is the head ganglion for the exteroceptive receptors and neurones, so the cerebellum is the head ganglion for the proprio-ceptive system. It is through enormous increase of the neural connections built on the distance receptors, that the development of higher animals is made possible; their domination over environment is secured not by increasing refinement of receptors, but by the elaboration of inner cerebral adjustments.

The final lecture deals with the question whether *sensory* coördination and fusion is to be interpreted according to the rules holding for motor coördination. The problem was studied experimentally on two retinæ; it was found that they must be held to retain independent function, and that they seem also to exhibit mutual interference. Hence the fusion of binocular images seems to be a fusion of already elaborated perceptual products. The convergence of paths from corresponding retinal points to the same cortical area is regarded, not as a basis for sensory fusion, but as serving for the unified efferent discharge by which the contraction of the eye muscles is regulated. In the final sections the relations between psychology and physiology are noted.

NEUROSES.

NEURASTHENIA.

Etiology and Symptoms. Charles D. Cleghorn[1] analyzes six thousand cases of neurasthenia out of 37,564 patients who have applied for treatment in the neurologic department of Vanderbilt Clinic during the eighteen years from October, 1888, to November, 1906. No cases showing admixture of hysterical symptoms or insanity were admitted to consideration. Of the 6,000 cases 58.6 per cent. were males and 41.4 per cent were females. As to age 6.7 per cent were from to 10 to 20 years of age, 39.4

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(1) Med. Record, April 27, 1907.

per cent. from 20 to 30, 30.3 per cent. from 30 to 40, 14.2 per cent. from 40 to 50, and 8.9 per cent. from 50 years upward. As to occupation, little can be concluded. The figures follow:

OCCUPATION.	No.	OCCUPATION.	No.
Housework	542	Salespersons	92
Clerks	340	Agents	90
Laborers	297	Porters	85
Tailors	286	Printers	86
R. R. Workers	188	Tobacco Workers	79
Factory hands	174	Painters	73
Drivers	156	Barbers	61
Domestics	142	Plumbers	58
Sewing	137	Peddlers	56
Storekeepers	136	Students	51
Operators	117	Watchmen	28
Carpenters	106	Weavers	26
Waiters	103	Firemen	23
Metal Workers	94	Teachers	19

A definite *etiologic* factor was recorded in 1,793 cases, overwork, worry, alcoholism, excessive use of tea or coffee, illness, shock, and trauma being oftenest mentioned. The *symptoms* are set forth in the following table:

SYMPTOMS.	No.	Per C't.	SYMPTOMS.	No.	Per C't.
Weakness	742	12	Lost taste, smell	5	..
Tremors	1087	18	Poor Appetite	898	15
Twitching	166	2	Constipation	1499	25
Reflexes+	340	5	Diarrhea	90	1
Reflexes —	54	1	Flatulence	387	6
Paresthesia	434	7	Pain in Stomach	297	5
Hyperesthesia	8	..	Palpitation	475	8
Fatigue	242	4	Pain in Heart	103	2
Headache, Occip.*	860	14	Vasomotor disorder	429	7
“ Frontal	672	11	No capacity work	197	3
“ Vertical	295	5	Anemia	529	8
“ Temporal	107	2	Secretory disorder	46	..
Dizziness	560	9	Poor memory	338	5

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*Patients whose symptoms included constipation and headache with unrecorded location were put in Frontal Headache class.

Heavy head	60	1	Worry	444	7
Backache	518	8	Fears	351	6
Tenderness	12	..	Emotional	550	9
Vague feelings	705	12	Insomnia	1684	28
Tired Eyes	22	..	Drowsiness	78	1
Poor Sight	83	1	Depressed	599	10
Poor Hearing	20	..	Syphilis	199	3
Tinnitus	101	2			

To summarize: "1. Neurasthenia is much more frequent in men than in women. 2. It occurs between the ages of 20 and 40 in two-thirds of the cases, few comparatively starting after 40. 3. The indoor occupations furnish a large majority of all cases. 4. Among causative factors, the disturbances of the gastrointestinal tract and the intoxications stand out as principals. 5. The most constantly present symptoms seem to be headache, insomnia, and constipation."

Tropical Neurasthenia is discussed by Louis H. Fales,[1] who has had some experience in the Philippine Islands. The literature on this subject is rather meager, yet nearly all American women and a large proportion of American men who have been in the Islands one year or more suffer, at least to some extent, with nerve exhaustion. "There is no doubt that directly and indirectly tropical neurasthenia incapacitates more white men in the Philippines than all other causes combined." Under symptomatology of this affection, Fales points out the following minor differences between it and neurasthenia as seen in the United States:

"1. In regard to women, menstrual derangements, metrorrhagia, menorrhagia, and dysmenorrhea are much more prevalent in the Philippines as an accompaniment of neurasthenia than in this country.

"2. Vasomotor symptoms seem to be somewhat more prominent in tropical neurasthenia.

"3. Tropical neurasthenia responds more readily to treatment than ordinary neurasthenia. Change of climate is especially effective. Many subjects leave Manila in a most exhausted condition, but after a short stay in Japan or a voyage across the Pacific, their condition is remarkably improved."

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(1) Am. Jour. Med. Sciences, April, 1907.

The cause for the condition is found chiefly in the direct effect of the actinic rays of the sun, and in the effect of continuous and excessive moist heat. There are but few localities in the United States which surpass Manila in relative humidity and none where the heat is so great. This leads to an abnormal dilation of the cutaneous capillaries and a consequent ischemia of the deep lying organs. Hence vertigo and faintness are very common, especially among women. Moreover, large men with relatively less skin-surface, are less able to resist the heat than men of smaller stature.

Blondes, who are more susceptible to actinic rays than brunettes; large men with great musculature; those with tuberculous tendencies; those having disorders of the stomach and intestines; those suffering from headaches, insomnia, hysteria, etc.; women, children, and old men, should avoid the tropics. The following rules of tropical hygiene should be observed:

1. The avoidance of the direct rays of the sun, chiefly to escape the actinic rays, by exposing one's self to the sun as little as possible; and by employing proper protection and clothing. The midday siesta should not be forgotten.
2. The avoidance of excessive heat by leaving the Islands during May and June.
3. Care in the selection and preparation of the food. It is a mistaken notion that dwellers in the tropics need little meat. More meat is needed than in our temperate climate. All food must be aseptically prepared, no vegetables eaten uncooked, no water swallowed unboiled.
4. The avoidance of excessive labor and long hours. Manual labor in the sun is out of the question. Light manual labor in airy shops is less depressing, but should be limited to five hours daily. Mental work is also much more fatiguing than in America, and prolonged concentration of the mind almost impossible.

Treatment. Alfred Gordon[1] discusses the rôle of organic phosphorus in the treatment of asthenic conditions in various nervous diseases. He characterizes asthenia as a deficient resistance to morbid influences in both the motor and psychic spheres. Phosphorus assists in the growth of

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(1) Medicine, December, 1906.

the organism, and in its recuperation. Dogs deprived of phosphorus quickly lose weight and become exhausted. Careful observers are cited by Gordon to prove that it is not phosphoric acid but organic combinations of phosphorus which are absorbed and utilized by the organism. In 56 cases of asthenia in nervous diseases, with a pronounced sense of exhaustion, phosphorus in organic form was made use of. In eight post-grippal neurasthenics the favorable effect of the phosphorus was unusually marked. In six other neurasthenics the results were also excellent. "As to the question of the relative value of the various organic combinations of phosphorus, the glycerophosphates appear to give the best results."

HYSTERIA.

Hysteria in Children. D'Orsay Hecht[1] reports five cases of hysteria in children and discusses the general subject. As to etiology, the chief predisposing factor is heredity. Sometimes hysterical symptoms are engrafted on those of organic disease, and outlast the latter. Sometimes a child's aptitude for imitation may lead to marked degrees of mimicry. Thus it is that hysterical parents have hysterical children, in many instances.

Symptomatology. Juvenile hysteria is chiefly monosymptomatic. In the adult the hysterical syndrome is seldom complete without stigmata and accidents in great numbers. In an hysterical child a single hysterical symptom stands out in bold relief, and should, from its very prominence, arouse a suspicion as to its functional nature. The paralyses of hysteria in children are chiefly monoplegias and paraplegias; hemiplegia occurs, but always without involvement of the face. The monoplegias usually appear with apoplectiform suddenness, are characterized by flaccidity, normal tendon reflexes, and in long-standing cases by slight degrees of atrophy from disuse. Some authors assert that hysterical anesthesia is as common in children as in adults, but Hecht says they are rarely noted, and that hemi-anesthesias are practically unknown, except in children of more advanced years. Hysterical motor agitation occurs frequently in children as choreic movements,

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(1) Jour. Am. Med. Assoc., Feb. 23, 1907.

a variety of facial habit spasms, convulsive tics, and epileptoid seizures. Grave forms of hysterical convulsions rarely occur in children.

Diagnosis. Symptoms may be regarded as hysterical which can be voluntarily produced. This at once prohibits any of the following from being classed as functional: the lost knee-jerk, the Babinski toe-sign, the reaction of degeneration, choked disc, optic atrophy, unilateral vocal cord paralysis, incontinence of urine and feces, facial paralysis, hemianopia, decubitus, ankle-clonus, and marked pupillary changes. Very important in leading to a proper diagnosis is an examination of the child's temperament. Since the hysterical child is apt to exaggerate every detail of its trouble, it is well to note any disproportion between cause and effect. Slight cause and grave consequence foreshadow hysteria.

Treatment. All hysterical symptoms are psychic, and the means of cure must be psychic. Just as hysterical symptoms are ushered in on slight provocation, so they may be made to disappear with slight but appropriate treatment. The parents must be made to understand the situation, and to show a dispassionate interest, or no interest at all in the child. Since so few parents will treat their hysterical children properly, isolation is often required. Strange surroundings, strange people, and a strange physician may work wonders. Psychically there are two methods at our disposal, the method of surprise and the method of disregard. The child may be surprised into finding suddenly, for example, that he can stand when he thought he could not, and if the physician grasps the psychologic moment he can effect a cure then and there. Better is the method of studied disregard of the child's complaints. Besides these measures, massage, electricity, the lukewarm bath and cold spinal douche or cold drip sheet are of use. Hydrotherapy and electrotherapy, being unpleasant and in part painful, are hence successful.

Hysterical Fugues. Hugh T. Patrick,[1] in his presidential address before the American Neurological Association on ambulatory automatism, reports six cases, and gives an extensive résumé of the literature of the subject. Owing

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(1) Jour. Nerv. and Ment. Dis., June, 1907.

to the fact that Charcot's first case was, by him, ascribed to epilepsy, it has since been the fashion to attribute most of such cases to that disease. As a matter of fact, however, some of them are undoubtedly manifestations of hysteria, and some of mere degeneracy. Of Patrick's six cases not one should be ascribed to epilepsy.

To Patrick's cases of ambulatory automatism may be added another reported by Claude and Rose(1) in a woman 25 years of age. In this case, too, despite attacks of loss of consciousness, without convulsive movements, however, the authors ascribe the fugues to hysteria.

Hysteria of the Intestinal Tract. Abel Gy(2) discusses hysteria as it affects the intestinal tract, ably and thoroughly. The symptoms are very diversified, and include enteralgia, hemorrhages, diarrhea, constipation, followed by intestinal obstruction and fecal vomiting in rare instances. In another group of cases gas produces tympanites which may be generalized, simulating pregnancy, or acute or chronic general peritonitis; or it may be localized and simulate a tumor or appendicitis. All of these conditions are described as to symptoms, and the diagnosis elaborated.

Paralytic Ptosis. This condition is said by C. Sauvineau(3) to occur in hysteria, and he reports two illustrative cases, the first showing true bilateral ptoses, the second showing a ptosis on one side accompanied by a blepharospasm on the opposite side.

Hysterical Joints. L. F. Barker(4) discusses the arthralgias of hysteria, traumatic neurosis, neurasthenia and psychasthenia. "The knee is most often affected (gonalgia), the hip next (coxalgia), the shoulder frequently, the talocrural and the intertarsal joints occasionally. The cervical spine may be the seat of symptoms and the condition be mistaken for cervical caries. One joint is usually affected at a time, though in rare instances two or more are concerned. Redness, heat and swelling are rarely present; pain and disturbance of function are the two prominent symptoms. Spasm (contracture) of the muscles proximal and distal to the joint frequently exists. The muscular atrophy is generally slight in amount, but in long-standing cases may be profound. The position assumed by the limb may or may not simulate that found in organic disease.

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(1) Rev. neurol., Feb. 28, 1907, p. 181.
(2) Gaz. des Hôpit., March 23, 1907.
(3) Rev. neurol., Feb. 15, 1907.
(4) Jour. Am. Med. Assoc., Feb. 2, 1907.

"Many of the cases follow trauma of some sort (Charcot). The trauma may be followed immediately by the joint neurosis; more often there is an incubation period of psychic meditation. In some cases no preceding physical trauma can be made out, the malady then developing after an emotional shock, after a convulsive seizure, or by imitation after seeing an organic joint affection. The arthralgia is sometimes the first symptom of the psychoneurosis; as a rule, however, the stigmata of hysteria, of psychasthenia or of neurasthenia have been recognizable, in cases carefully studied, for some time preceding the joint trouble.

"The great importance of the *differential diagnosis* of these arthralgias from organic disease, especially from tubercular gonitis and coxitis, is obvious. The nocturnal exacerbations of the pain, which frequently occur in sleep and awaken the patient in tubercular coxitis, are absent in psychoneurotic coxalgia. On making passive or active movement of the joints in cases of joint neurosis, the patients complain of 'horrible, unbearable pain'; sympathy only increases the pain, while if the attention can be diverted temporarily the movements can often be carried out without severe pain. It is often found, further, that the pain is more intense in the skin and soft parts than in the joints themselves. Careful esthesiometric tests nearly always show a distinct hyperalgesia of the skin over the joints between the attacks of pain. In hysterical coxalgia, for example, there exists frequently an area of cutaneous hyperalgesia, triangular in shape, the apex of the triangle being at the lower margin of the symphysis pubis, the base at the middle of the sacrum. When the knee, elbow or wrist are complained of, the hyperalgesia of the skin is usually cuff-like in form, the area surrounding the whole joint. During the severer neuralgiform seizures the hyperalgesia may change to a total hyperalgesia of one-half of the body. Analgesias are sometimes demonstrable in hysterical cases.

"In psychoneurotic coxalgia the attitude of the lower extremity may resemble most closely that met in organic disease of the hip joint; there may be apparent shortening, abduction, lateral rotation, or, later on, adduction and medial rotation. The limping gait, accompanied by lumbodorsal pseudoscoliosis, may strikingly simulate that of organic hip disease. Some of these patients refuse after a time to make any attempts to move their joints, and lie in bed for months or even for years.

"With x-ray examinations and tuberculin reactions organic arthropathies are now less likely than formerly to be mistaken for joint neuroses, but there is still danger that the psychoneurotic joint will be looked on as an organic disorder. In doubtful cases deep chloroform narcosis should be resorted to for diagnostic purposes. Not only do the contractures disappear when the patient is anesthetized, but a careful physical examination of the joint can be made and the physician can assure himself of objective negativity. In addition the phenomena observable during recovery from anesthesia are helpful in differential diagnosis. It is the sensitiveness of the skin which returns first in the psychoneurotic joint, the tenderness and pain in the deeper parts, elicited by tapping the trochanter or the heel, coming back later. It is only after the patient has rather fully recovered from the anesthesia, say in the course of from twenty to thirty minutes, that the deep pain and contractures return. It is just the opposite with the joints of organic disease.

"The *treatment* of the psychoneurotic arthralgias should be mainly directed toward the general psychoneurosis which underlies the symptom. Nothing is more harmful than a predominantly local therapy. Isolation and psychotherapy are the sovereign remedies in these cases. After a thorough examination has been made and the absence of organic disease has been determined, the patient should be told that the joint trouble is nervous in origin, and that, in the physician's opinion, there is no reason why it should not speedily get well. After a few days of complete separation from the family and friends, and when 'medical obedience' has been fairly well established (frequently easily obtainable by encouragement and by keeping the patient for a short time in bed on a diet consisting exclusively of milk), the patient's intellect and will are to be appealed to by 'persuasion.' Passive movements of the joints are to be begun and gradually increased; hydrotherapy and electrotherapy may be used as adjuvants. After a few days the patient may be induced voluntarily to move the joints a little, and very soon normal motility may be regained. The patient, through occupation therapy and will-gymnastics, should be taught gradually to improve in self-control."

Treatment of Hysteria. The treatment of hysteria is set forth by Thomas D. Savill.[1] Study of the disorder has led him to conclude that: 1. Hysteria consists of an instability or undue irritability of all the nervous and reflex centers throughout the body, and particularly those of the vaso-motor and sympathetic systems. 2. Hysterical paralysis or tremor and many other hysterical phenomena are produced by vascular changes in the nervous system or elsewhere. 3. The essential defect on which hysteria depends is inborn and inherent.

Savill's remarks on the treatment of hysteria come under five headings: (*a*) Certain general measures for improving the tone and nutrition of the nervous system and to allay its irritability; (*b*) other symptomatic treatment, mainly by the aid of drugs; (*c*) etiologic treatment which depends on the discovery and eradication of the exciting causes; (*d*) the use of vaso-constrictors, vaso-dilators, and blood remedies; and (*e*) educational and other preventive measures.

(*a*) The general measures directed to the improvement of the nervous irritability are seven in number: (1) Complete *rest* of the body and mind is imperative, and can only be obtained in bed. Abundance of sleep is indispensable, even if hypnotics must be given to produce it. (2) *Isolation* from home surroundings is almost as important as rest, for it not only relieves the patients at once from worries incidental to their surroundings, but it attracts their attention into new channels and away from their disorder. Even letters to be written or received should be interdicted. (3) As much easily *assimilable food* as the patient can take should be given; and in order to increase the power of assimilation, (4) general *massage* should be ordered. The combination of these four measures, rest, isolation, abundance of food, and general massage, constitute the Weir-Mitchell treatment. (5) *Electricity* is of considerable value, in any form. Galvanic and faradic currents are very useful for hysterical affections of the voluntary muscles. The constant current is more useful for rigidities, the faradic for paralytic conditions. Static electricity combines three effects. It undoubtedly produces a psychic effect on the patient; it also seems to improve his general tone; and lastly it appears to be of special value in cases of anesthesia. (6) *Hydrotherapy* is not much employed in England, yet cold or hot water applied to the surface of the body either by douches or baths acts powerfully on the vaso-motor system. (7) *Psychotherapy* includes all kinds of persuasion, suggestion (auto- and hetero-) and hypnotism, and they may be valuable. The object of psychotherapy is to cultivate the will and the control of the patient over unruly emotions and unstable reflex centers. Hypnotism cannot be used on many. Auto-suggestion is of great value.

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(1) Lancet, June 22, 1907.

(*b*) Other symptomatic treatment mainly with the aid of drugs. As to sedatives, the bromids remain our sheet anchor, and the ammonium salt is the best. Opium and its alkaloids are to be prohibited, first because they may lead to a habit, and secondly because they do not always act as sedatives. Apomorphin hypodermically is the most valuable remedy we have for the prompt cure of severe and recurrent hysterical convulsions. Alcoholic stimulants are forbidden. Valerian and asafetida are valuable stimulants for faintings, prostration, and collapse.

(*c*) Etiologic treatment must be based on a careful study of the accidental or determining causes of the hysterical manifestation. In a series of six cases reported Savill illustrates cures based on such study, he having come to the conclusion that the chief seat of the reflex instability lies in the sympathetic vaso-motor system. He therefore determined to look out for, and if possible correct, any derangement of metabolism, blood alteration, or constitutional disorder prevailing. The results have been gratifying when such defects, if found, have been corrected.

(*d*) During the last few years Savill has used vaso-constrictors (e. g., ergot), vaso-dilators (e. g., pilocarpin), and remedies acting on the blood (e. g., calcium chlorid) in cases of hysteria of various kinds showing irregularity in the vaso-motor regulator mechanism, or a suspicion of dilatation or constriction of the vessels of the central nervous system. A case is reported in illustration. As yet Savill is unwilling to generalize, his cases thus treated having been too few.

(*e*) Educational and other prophylactic measures are of great value. The cultivation of the will and of self-control is the central point in the education of the hysterical. "As age advances any tendency to introspection should, as far as possible, be quietly corrected, and especially by the cultivation of interests outside self. But, in guiding these people towards such counter attractions, screen them, as far as possible, from undue religious exercises and love affairs—two fatal directions which their fancies are very apt to take. On the other hand, the cultivation of the gentler qualities, pity, self-sacrifice, and work for others, leads to the formation of character and the self-control we desire."

EPILEPSY.

Symptoms. N. B. Ross[1] reports a case of unusually *low temperature* following status epilepticus, in a boy 10½ years of age under treatment at the Sonyea Colony for 40 days. The boy's last illness began July 25, 1905, with a temperature of 99° F., rising to 103° F. in two days. The temperature fell slowly till August 1, and then it began to fall rapidly, reaching a minimum of 96.4° F. on that day. At 2 a. m. August 2 the temperature was 95.2° F.; at noon it was 86° F.; at 8 p. m. it was 81° F. Death took place at 10:35 p. m. In each of the last three records the temperature was taken by rectum, the thermometer being left in place 20 minutes.

H. Claude[2] reports a case of epilepsy in which the aura was an "illusion of false consciousness," a *paramnesic aura* in a girl 20 years of age.

W. T. Shanahan[3] reports a case of epilepsy *associated with acromegaly,* in a woman 31 years of age. Similar cases have been previously reported. (PRACTICAL MEDICINE SERIES, Vol. X, 1905.)

Diagnosis. Frank M. Stiles[4] insists on early diagnosis in epilepsy. In answer to the question as to whether it is

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(1) American Med., October, 1906.
(2) Rev. neurol., Feb. 28, 1907, p. 188.
(3) Jour. Nerv. and Ment. Diseases, May, 1907.
(4) Med. Record, Dec. 8, 1906.

possible to say in any case that a primary seizure is epileptic, there are two factors demanding consideration: First, it is essentially a disease of early life, and secondly, an hereditary predisposition is found in a majority of cases. Next in importance to these two main factors is a third—that of the presence of mental or physical stigmata of degeneration. The author's conclusions follow:

"1. A diagnosis of epilepsy at the earliest possible stage of its development is essential to a successful treatment.

"2. Convulsions from any cause at any age are not to be regarded as of slight significance, but the possibility of the development of epilepsy in every case must receive careful consideration.

"3. Recurring convulsions in the young, associated with hereditary susceptibility to nervous diseases, should invariably be regarded as probably epileptic in their nature. The more insignificant the exciting cause the stronger the indication of this disease should be regarded.

"4. In all cases in which obscure mental or nervous symptoms tend to recur periodically with normal intervals the possibility or probabiilty of epilepsy in the form of psychic, Jacksonian, or petit mal seizures should be considered, and careful study be given to the symptoms; and when possible, the case should be placed under the care of an intelligent attendant until the diagnosis is positive.

"5. Though care should be exercised in doubtful cases it is better to give the patient the benefit of the doubt and institute treatment early, even when a positive diagnosis of epilepsy is not possible, than to allow the condition to become chronic and perhaps hopeless. A positive and early diagnosis is important, but the early treatment is of far greater importance, and proper treatment should be begun as soon as the diagnosis is reasonably certain and should be persistently carried out until the result is assured."

Treatment. *Medical.* Alfred Gordon(1) gives the results of the salt-free treatment of epilepsy apropos of thirty-seven cases. In order to determine the value of dechlorization, every one of the 25 adults and 12 children comprising the 37 cases was treated in an identical manner. Nitrogenous food and stimulants were absolutely forbidden, and the quantity of starchy food reduced to a minimum. Milk, water, eggs, some vegetables, fruit, and bread were the only articles allowed. Bromids were given uniformly with some variations according to the age of the patient. As soon as the number of seizures was markedly diminished, observations on the withdrawal of salt were made. The major attacks became less severe, shorter in duration, and at longer intervals. He concludes from this study that sodium chlorid undoubtedly plays an important rôle in the chemistry of the organism. The suppression of alimentary salt in the diet of epileptics has a favorable effect on epileptic seizures inasmuch as it reduces their frequency and their severity. It is of the same value in the treatment of epilepsy as the strict observance of dietetic and hygienic rules. Reduction or complete removal of sodium chlorid from the diet gives a better result while bromids are taken than while they are not taken. Dechlorization is certainly not a specific for epilepsy, but it adds a new element to our meager neurologic armamentarium.

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(1) New York Med. Jour., Oct. 20, 1906.

J. M. Bennion[1], from extensive observations on the treatment of epilepsy in the insane with strontium bromid, concludes as follows: 1. Strontium bromid, as a rule, acts better in controlling the number and severity of the fits than the mixed bromids of sodium and potassium. 2. Strontium bromid rarely causes depression. 3. Rashes remained absent in all cases thus treated. 4. Strontium bromid acted best and most consistently on the female patients, in all of whom the action of this drug, as regards controlling the fits, was far superior to that of the mixed bromids. 5. The maniacal symptoms of the male patients while taking strontium bromid were not influenced to any perceptible degree. 6. The mental condition of the female patients appeared to improve.

Operative. Concerning operation in epilepsy, T. H. Weisenburg[2] may be quoted as follows:

"No one questions the advisability of operating in a case of epilepsy which is due to a definite cause, as an injury, hemorrhage, or tumor, but to operate in cases of idiopathic epilepsy which have no discoverable focal basis

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(1) Lancet, Jan. 5, 1907.
(2) New York Med. Jour., Oct. 13, 1906.

either in the brain or in any other portion of the body, is a questionable procedure. Idiopathic epilepsy is an organic disease and is dependent on structural developmental faults. These imperfections are not located in any one portion of the brain, as the motor area, but probably involve all parts. The epileptic convulsion is only a symptom of a general disease. How futile, then, any focal brain operation!

"Extracranial operations have been done and are still practiced on the theory that toxins and other reflex causes occasion epilepsy. The surgical correction of ocular defects and the removal of nasal polyps may be an advisable procedure, but to ascribe these and similar defects as the causes of epilepsy is the veriest nonsense. Toxins unquestionably enter into the etiology of the convulsions as a symptom; but there is no reason to believe that toxins themselves are the ultimate causes of epilepsy."

M. Allen Starr,[1] in discussing the present status of brain surgery, calls attention to the futility of operations for epilepsy. True Jacksonian epilepsy, in which the diseased focus can be accurately localized, and in which it is invariably cortical and therefore within the operator's reach, is the only form of epilepsy which is open to operation. In Starr's experience not more than 2 per cent. of the cases of epilepsy are operable, the other 98 per cent. presenting none of the characteristic symptoms of localized disease of the brain. It is useless to trephine in idiopathic epilepsy, even though the condition apparently is due to a fall or blow on the head, unless the attacks are Jacksonian in type. Moreover, even in focal epilepsy, the prognosis after operation is not so favorable as was at first anticipated; the resulting cicatrix itself is an adequate focus for the recurrence of the epilepsy. In only about 20 per cent. of such patients operated on has permanent cure been the terminal result.

"Such unfavorable results might possibly lead to the conclusion that in the majority of these cases of local epilepsy an operation should be refused. As a matter of fact, however, it is often found that the focal epilepsy is the first symptom of the growth of a tumor, or the development of a cyst in the center of the brain. It seems imperative, therefore, to operate in these cases of focal epilepsy as

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(1) Jour. Am. Med. Assoc., Sept. 22, 1906.

soon as the diagnosis is made: first, because of the possible relief of epilepsy, and secondly, for the possible discovery of a more serious and fatal disease just beginning. I have had operations performed for the relief of epilepsy of a focal nature in about 60 cases. In a few of these cases conditions have been met at the operation which rendered any relief impossible. In a few of the cases (about one-fifth), a permanent cure has been obtained. In the remainder the epilepsy has returned and has been in no way affected by the operation. I have seen many cases of ordinary idiopathic epilepsy, supposed to be due to a blow on the head, operated on, the blow having left some scar that has guided the surgeon. I have never seen in these cases any tangible disease in the brain when it was exposed, and I have never seen any improvement or relief from epileptic attacks. A review of this experience convinces me that the operation of trephining in epilepsy is of very limited application, and is only to be recommended in a few selected cases which present the necessary guide to both physician and surgeon."

Institutional. Hugh T. Patrick[1], in an address before the Eleventh Annual Illinois Conference of Charities, Oct. 11, 1906, discusses the proper institutional treatment and care of the epileptic, and attempts to answer three questions: 1. What is the epileptic's condition? 2. What is being done for him? 3. What can be done for him? Of the 7,000 epileptics in Illinois only a very few have means ample enough to secure proper care. A great many of the 1,200 now in the asylums and poor-houses of Illinois are hopelessly insane or demented and need only kindness and custodial care. About 5 per cent. are cured by medical treatment, and more, a very indefinite number, if not cured, are relieved to such an extent that the disease constitutes no real disability. About 5,000 remain in Illinois to be dealt with, and their state is deplorable. "Imagine no profession, no business, no position of trust, no real occupation; no normal social intercourse, no steady purpose, no aim but to escape observation and to be let alone; no normal recreation, never any real fun; finally, no home of your own, no wife, no husband, no children—but the normal capacity to

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(1) Illinois Med. Jour., March, 1907.

appreciate and enjoy all of these blessings and the normal longing for them."

What is being done for the epileptic? Physicians are striving to help them, but there are two difficulties in the way: the first is the inherent difficulty of the disease; the second, the impossibility of having all their directions carefully and continuously carried out. Hence little is being accomplished. What can be done? "We can protect the community from the numerous calamities arising from epilepsy, and, to a great extent, we can protect the patient from his disease. We can protect the state against propagation by confirmed and hereditary epileptics, which would mean not only fewer epileptics in the future, but fewer criminals, fewer degenerates, fewer persons on public charge. * * * We can give the epileptic associates, associations, and recreation. We can give him vastly better health, radically cure some of him, and eliminate the feeling that he is an outcast, something apart—to be looked at askance."

These things can be accomplished by a liberally conceived, properly founded, well organized, scientifically conducted state colony for epileptics. "Our predecessors have not only shown the feasibility of the colony plan, but have worked out many details as well." * * * It may by some be thought to be an unkindness to put many epileptics together. * * * Quite the reverse is the case. Every asylum physician knows that epileptic patients naturally gravitate together. * * * To be an epileptic in such a colony is no more strange than to be an Eskimo in an Arctic village. In conclusion, then, there is no single valid objection to a state colony for epileptics, while good statesmanship, good citizenship, public policy and private duty, the love of God and the brotherhood of man all point to it as the best solution of the desperate problem of the epileptic."

MIGRAINE.

Differential Diagnosis. Sir William Gowers[1], in a lecture on migraine, its premonitory symptoms, relation to epilepsy, course, etc., points out the following differences between migraine and epilepsy:

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(1) British Med. Jour., Dec. 8, 1906.

"Their antecedents differ. When heredity is to be traced in epilepsy, the antecedents present either the same disease or insanity. These are seldom to be heard of in the case of migraine, in the more marked forms of which parental or ancestral gout is seldom absent, and generally is pronounced; and there is often also a history of severe headaches or neuralgia. The difference in the duration of the premonitory sensory symptoms is especially important. The brief warning of the epileptic fit, lasting only a few seconds, is not met with before pure migraine, and to this the deliberate disturbance, lasting from ten to twenty or thirty minutes, is confined. Rarely is it over in five minutes, but in such cases the same prodroma on other occasions endures for a much longer time, and the variations indicate its nature.

"The character of the forerunning disturbance is generally also distinctive. In the arm, the zone of tingling, slowly passing up the limb, is never described in epilepsy, of which any clonic spasm is distinctive, and unknown in migraine. The visual symptoms which so often precede migraine similarly differ in duration from those that constitute the warning in epilepsy, and they usually differ also in character. Dimness or loss of sight is general in epilepsy, and is momentary; in migraine it is blotchy and irregular, or one-sided and prolonged. A more definite sensation has a mechanical elaboration, and consists of an angled line, simple or multiple, straight, curved, or expanding, which may limit an area of loss.

"A stellate body, still or moving, may occur in either affection, but the simple flash of light, or multiple momentary bright 'stars,' so common in epilepsy, are not known in migraine. In this, also, we never meet with the elaborate psychovisual sensation—a face, a figure, a scene, or the like, which are not rare in epilepsy. The contrast is curious between this form of elaboration and that which is so common in migraine, which I have called 'mechanical'; it suggests that the latter is due to a disturbance in a region of the cortex that is at a lower level of function than that in which the epileptic process occurs, or that the mode of disturbance is quite different.

"The special characters of the pain are also important. The severe degree and long duration common in migraine are never met with after an epileptic attack of minor character, with which alone the symptoms of migraine can be confused, nor is severe headache, even after a convulsion, one-sided. Vomiting occurs soon after the fit in epilepsy, not after the headache has lasted some hours, as in migraine."

SYDENHAM'S CHOREA (CHOREA MINOR.)

Symptoms. W. S. Thayer[1] analyzes 808 cases of chorea treated at the Johns Hopkins Dispensary and Hospital, with special reference to the cardiovascular manifestations. Of the 808 cases 783 were in whites, or 96.9 per cent.; 25 or 3 per cent. only were colored. (The colored patients ordinarily average 12 per cent. of all.) Thus Weir Mitchell's observation that chorea is relatively rare in the negro is confirmed. As to age, 84.5 per cent. of the patients were between the ages of 5 and 15 years. In 62.4 per cent. the patient was seen in the first attack alone; in 20.4 per cent. there was a history of two attacks, and in 17.1 per cent. there was a history of three or more attacks.

As to rheumatism, 21.6 per cent. gave a history of this disease. Of 689 cases in which the heart was examined 235 or 40.5 per cent. showed murmurs. In about one-quarter of the cases the murmurs were soft systolic souffles, heard at the base alone, or over the whole cardiac area, and unassociated with other modifications of the size or functions of the heart. In 25.4 per cent. of cases in which the heart was examined, organic cardiac disease was found. Most of these lesions were those of mitral insufficiency, but many were combinations of mitral stenosis and mitral insufficiency, or mitral and aortic insufficiency.

Four cases were cases of *chorea insaniens* and all resulted fatally. Cardiac manifestations were more frequent in those patients who had suffered from acute polyarthritis. From the clinical symptoms and the condition of patients returning to the hospital subsequently, there is considerable reason to believe that, in a large proportion of these febrile cases, acute endocardial complications were present, especially if fever occurred independently of joint-complications.

A case of *unilateral paralytic chorea* is reported by

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(1) Jour. Am. Med. Assoc., Oct. 27, 1906.

Julius Grinker[1], who quotes Todd's Clinical Lectures on Paralysis, published in 1850, as follows:

"In a large proportion of cases of chorea the choreic movements occur more on one side than on the other, and sometimes they will be altogether confined to one side—the child being hemiplegically affected. In such a case, as happens when the choreic movements have wholly or partially subsided, the limbs that were previously twitching now become paralyzed. The case now resembles in every detail that of ordinary hemiplegia from cerebral causes. But the following points of difference can usually be observed: 1. The face is often not affected, or slightly so. 2. There will not be any paralysis of the tongue, but more or less of the peculiar mode of protrusion which characterizes chorea. 3. The paralyzed limbs will exhibit, even in a very slight degree, the choreic movements."

Grinker extends O. Förster's analysis of the motor disturbances of chorea. Förster divides them into (1) spontaneous choreic movements, and (2) incoördination of voluntary movements. Grinker subdivides the last heading into (a) incoördination proper, and (b) muscular weakness. He believes that the disturbances of coördination are due to a lack of promptness, and to instability of, muscle innervation. "The impulse is either delayed or side-tracked into byways instead of reaching the intended muscle paths. Or, if the intended movement is executed there are so many accessory irrelevant movements that an incoördinated act is the result, instead of the orderly coördinated intentional movement of health. Muscular weakness results from either a delayed nerve impulse or from dissipation of well-directed energy into channels having an inhibitive effect on the intended movement."

"To summarize this analysis, we have, on the one hand, the involuntary so-called choreic spontaneous movements which depend on an irritation of single muscles and are dissimilar to voluntary movements. On the other hand, we have disturbances of coördination; that is, a disturbance either in the execution of voluntary movements or in the maintenance of static equilibrium. The incoördination manifests itself in its complete form as follows: The principal muscles concerned in voluntary movements are promptly innervated, but the impulse is miscarried to a varying degree. In severe cases it is so ineffectual as to cause distinct paresis affecting equally all the muscles of an extremity. In the simple movements of the fingers, tongue, and lips the innervation may be delayed and intermittent.

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(1) Amer. Jour. Med. Sciences, November, 1907.

"The impulse of the so-called agonists, the muscles directly concerned in a movement, as opposed to the antagonists, which merely assist the agonists, may radiate into other channels or along the commissural tracts to the opposite side; then we have purposeless associated movements. In like manner the antagonists that support or maintain the agonists are not properly innervated in severe cases of chorea and we have intermittent muscular contractions. In a general way we may state that those impulses that are directly concerned in the volition of station or locomotion are functionating properly; in fact, there is even an excess of function leading to associated movements, while those involuntary motor impulses concerned in the maintenance of equilibrium which emanate from the cerebellum and its paths are markedly deficient in chorea."

The patient, a boy of seven years, after being kicked in the right ankle by a companion, slowly developed a right-sided hemiparesis, the weakness beginning with limping, and slowly involving the whole side except the face. The reflexes were slightly reduced on the affected side; there were no atrophies, spasticity, or sensory disturbances. When the boy tried to use his right hand and arm a peculiar jerkiness and tremor appeared, resembling partly the intention tremor of multiple sclerosis, and having some of the characteristics of tabetic incoördination. No hysterical stigmata could be found.

George E. Price[1] writes of an unusual symptom in chorea, viz., *excessive secretion of saliva,* accompanied by drooling. This symptom has been observed not infrequently in conditions characterized by marked motor disturbance, such as hydrophobia and paralysis agitans. Price reports three cases of chorea in which the symptom was present. He says that in each instance the patient had one attack of

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(1) New York Med. Jour., April 27, 1907.

chorea prior to the attack observed, that each case was a severe one, and that two of the three showed valvular defects of the heart. He believes the symptom to be purely mechanical in origin, due to excessive movements of the tongue and muscles of mastication exciting the salivary glands to increased activity, while at the same time the involvement of the tongue, jaw, palate, and pharynx markedly interferes with swallowing. Hence the drooling.

A. Charpentier[1] describes a patient, 12 years old, suffering with Sydenham's chorea following acute articular rheumatism. The patellar jerks and the Achilles jerks were both equal on the two sides and normal; but *Babinski's sign* was very plainly evident in the left great toe. This would seem to indicate some perturbation of the pyramidal tract fibers in chorea minor.

Chorea Insaniens. J. Magee Finny[2] reports in detail a case of chorea insaniens in a girl of seventeen years, terminating fatally in nine days. Clinically the patient showed a most severe chorea which immediately followed rheumatic pains in the legs. When the pain ceased the movements began, and almost at once the patient complained that her mind was being affected. The choreic movements were so violent that she barked her elbows, rubbed her heels sore against the bed-clothes, with the heel of one foot abraded the skin of the opposite instep, and stripped the skin from over the sacrum. She could hardly speak, but at times was calm for a few minutes, and could then be fed with soft thick food. The rectum and bladder were emptied involuntarily. Owing to the involuntary biting by her teeth, and the rubbing of them by her hands, her lips were sore and bleeding. She was indeed a pitiable object. Chloral hydrate and chloroform inhalations were the only drugs capable of quieting her even temporarily. Rectal feeding was impossible. She was quite maniacal at times, striking out, shouting and screaming, or uttering deep raucous sounds, yet she had periods of calm in which she would say some words intelligently, such as: "No, thank you, sir." At one time there were hallucinations and delusions, and toward the end a period of semi-coma.

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(1) Rev. neurol., Dec. 15, 1906.

(2) Dublin. Jour. of Med. Science, May, 1907. Also, Brit. Med. Jour., April 27, 1907.

The postmortem examination showed the vessels of the cerebral cortex engorged. The brain was fixed in formalin and subsequently examined microscopically in various parts. In the Rolandic area some of the small cortical vessels were thrombosed, while in others, collections of cells with oval nuclei lay heaped up in the perivascular lymph-spaces, and there were clumps of what appeared to be broken up nuclear material in these spaces. The spinal cord showed nothing abnormal, and the cerebrospinal fluid was sterile.

Of all the grave varieties of chorea, maniacal chorea or chorea insaniens is the gravest as well as the most fatal. Of 23 cases collected from various sources by William Gay death occurred in 8, recovery in 10, permanent weakmindedness in 1, and 4 were lost sight of, having been sent to asylums. The duration may be very short, death coming even as early as the sixth day, and in one instance on the second day. As to etiology, Finny believes in the bacterial origin of the disease, notwithstanding the fact that no organism was found in his case.

Treatment. J. Babinski[1] has used scopolamin in chorea minor with happy results in certain cases. He has treated four patients with it, using subcutaneous injections of the hydrobromate of scopolamin in doses of 0.2 to 0.5 of a milligram daily, with the result that there has been a decided improvement. In one of the cases, of great intensity, antipyrin, arsenic and chloral were useless, the patient having grown progressively worse while taking them.

DISEASES OF THE BRAIN AND MENINGES.

CEREBROSPINAL MENINGITIS.

During the past year a very large number of articles have appeared upon epidemic cerebrospinal fever, owing to the large outbreaks in this country, and in the cities of Belfast, Leith, Glasgow, London, Hamburg, and some small German mining towns. Much work, too, has been done on the *Diplococcus intracellularis,* its identification, agglutination and opsonic reaction. Only a small number of the monographs are here reviewed.

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(1) Rev. neurol., Jan. 30, 1907, p. 86.

Mode of Infection. William Robertson[1] says that at the outbreak of the Leith epidemic he was struck with the fact that the victims of epidemic cerebrospinal meningitis were to be found solely among families of the casual or regular dock laborers. This feature was gradually lost when the epidemic became widespread. The case in favor of oversea conveyance of infection was made strong by this involvement of dock laborers.

"That foreigners coming to this country, either to stay here or on their way to America, had conveyed infection with them, was brought into striking prominence by the fact that a ship steward whose work brought him into intimate contact with these aliens developed epidemic cerebrospinal meningitis and died; that a railway clerk whose duties consisted in looking after transmigrants at one of the Leith railway stations also took ill and died; finally, that a 6-year-old daughter of a customs officer was seized with illness and succumbed. The employment of laborers at the docks is not regular, consequently groups of them stand about the quays or dock entrance waiting for work. Inside the docks smoking is strictly prohibited, chewing is accordingly resorted to, and spitting accompanies chewing. Should the sputum contain meningococci, they are readily blown about the extensive sheds which adjoin the quays. At the dock gates the idlers, or those looking for work, collect in more compact groups. Smoking and chewing is indulged in, and spitting is constant. Here also diffusion of meningococci becomes easy."

J. S. Fraser and John D. Comrie[2] were stimulated by the papers by Ludwig Jehle on the epidemiology of cerebrospinal meningitis, to investigate the recent epidemic of that disease in Leith, Scotland. Jehle's views[3] are best summarized in the accompanying tables. He points out that school epidemics of this disease are unknown; that second cases in the same house are uncommon; that cases of cerebrospinal meningitis placed in general wards give rise to no new cases. Jehle concludes that cerebrospinal meningitis is not spread by children as are other infectious diseases. On the other hand, coal mines are good incubators,

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(1) British Med. Jour., July 20, 1907.
(2) Scottish Med. and Surg. Jour., July, 1907.
(3) Wien. klin. Woch., 1906, No. 25, and 1907, No. 1; also Münch. med. Woch., 1906, No. 29.

being warm, dark, and moist. Miners also make good carriers of infection, being subject to chronic rhinitis and pharyngitis. On the return of the miner to his home his children kiss him, or use the same dishes as their father, or, as in the case of very young children, they may crawl about on the floor on which the miner habitually expectorates.

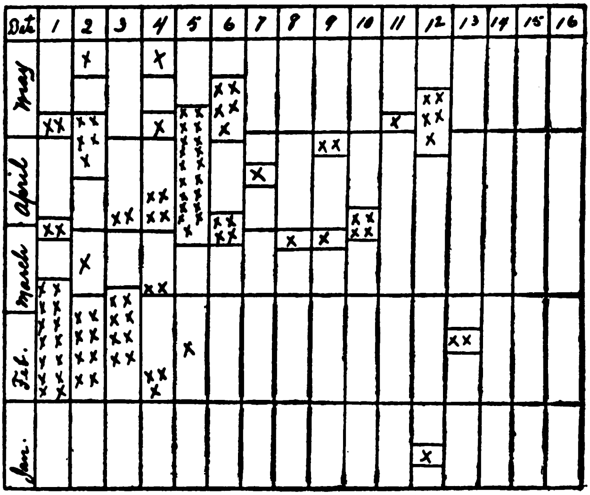

Fig. 1.—Orlau Karwies Mine. Each case is marked with an x.

"This figure shows the cases connected with the 16 shafts of one mine. It will be seen that very many cases occurred among the children of miners working in certain shafts, while those of the miners in other shafts were free from the disease. The families in which these cases occurred did not know one another, and had no connection with one another above ground. The connection is, however, quite clear when we remember the contact of the fathers in the mine shaft. From the facts indicated in the above figure Jehle has come to the conclusion that in mining districts the coal mines play the same part in spreading cerebrospinal meningitis as the schools in the spread of other infectious diseases."

Fraser and Comrie, with these ideas of Jehle fresh in mind, investigated thoroughly the Leith epidemic. There were 83 cases of cerebrospinal meningitis in Leith up to May 28, 1907, and 53 deaths. A map prepared showed the cases to be most numerous in the densely crowded and poorer parts of the town near the docks. Observation

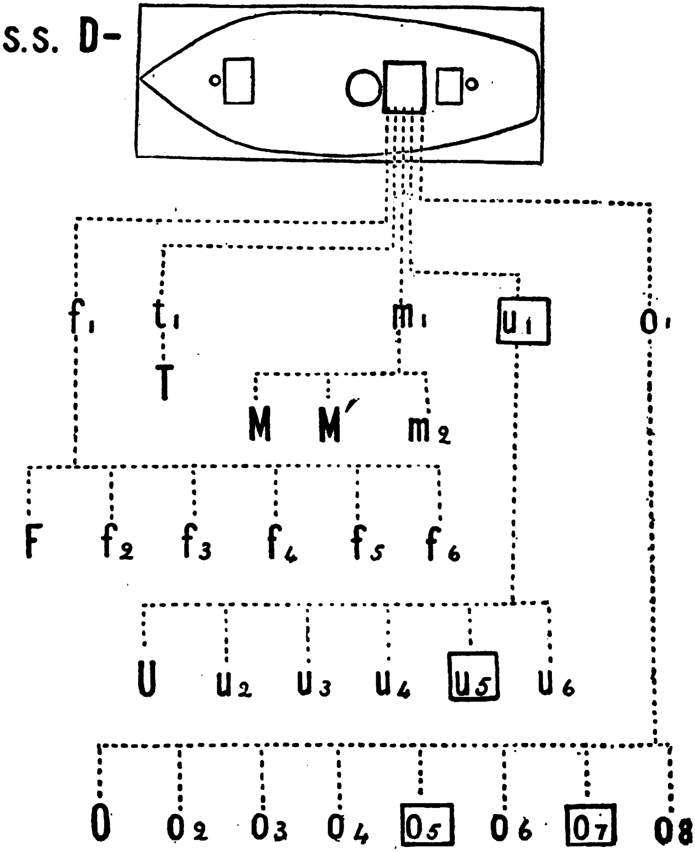

Fig. 2.—Diagram showing the relation of five affected families to the infected ship ss. D—— at Leith. The five fathers are indicated as f_1, t_1, m_1, u_1 and o_1, and the cases of meningitis in their families as F, T, M, M′, U and O. The letters surrounded by squares, u_1, u_5, o_5, o_7, indicate individuals in whose naso-pharynx the meningococcus was found; in the others it was sought for, but not discovered.

teaches that during this present year seaports have been specially affected, notably Glasgow, Belfast, Leith, New York, New Orleans, Boston, Hamburg, Copenhagen, and Antwerp. Inland cities also have been affected, but the number of seaports involved is certainly noteworthy. It is at least possible that emigrants play a part in spreading the infection, and in a ship below decks are conditions to some extent resembling those in a coal mine, darkness, moisture, and heat. The diagram (Fig. 2) shows how a certain steamer gave rise to several cases. The following conclusions are reached by the authors:

1. That hot, dusty, ill-ventilated atmospheres, which provide conditions favorable to the growth of the meningococcus and to the occurrence of the nasopharyngeal catarrh, are often associated with the dissemination of epidemic cerebrospinal meningitis.

2. That the high comparative percentage of fathers, whose nasopharynx was found to contain the meningococcus, points to the fact that they probably are the carriers of the disease to their children.

3. That the chief incidence of the disease in the Leith epidemic was among children of the lower classes, and that this is in favor of a nasopharyngeal infection.

4. That infection of the nasopharynx is undoubtedly an important factor in spreading the disease.

5. That it is advisable to isolate all contacts and to carry out a bacteriological examination of the nose and nasopharynx. If the meningococcus be present these cavities should be disinfected, and the contacts should only be allowed to leave quarantine after the nasopharynx has been reported free from the meningococcus on two consecutive occasions.

Study of the Diplococcus Intracellularis. Thomas Houston and J. C. Rankin[1] publish a note on the opsonic power of normal serum with reference to the diplococcus found in the Belfast epidemic. Normal serum was found to have an extremely low opsonic power, whereas the serum of many of the cerebrospinal cases had an exceedingly high opsonic one, so that in many of the preparations containing

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(1) Lancet, May 4, 1907.

the patients' sera the leucoytes were packed so full of cocci that accurate counts were impossible.

"In making the opsonic determinations in the usual way and incubating the mixture of washed corpuscles, emulsion of meningococci and serum, for 15 minutes, it was at once observed that many of the preparations containing the patient's serum showed marked agglutination of the meningococci, while the controls never showed any. In order further to test this absence of agglutination with non-infected serum a number of different normals (seven in all) were at different times used as controls, also sera from cases infected with tubercle, typhoid fever, staphylococcus and *Micrococcus melitensis,* as well as rabbit and guinea-pigs' sera. In no case was there any agglutination observed with such sera. In other words, the opsonic method in the case of meningococcus provides us with two distinct indications to determine the presence or absence of infection: first, the opsonic index; and second, the presence or absence of agglutination in the preparations."

The following deductions are drawn from 63 determinations of the opsonic power in cases of cerebrospinal fever in the Belfast epidemic:

1. From the sixth day on, all the cases examined (with one exception on the seventh day) showed an opsonic index of over 4; several of the cases from the second day onwards also gave an opsonic index of much above 4.

2. The opsonic index seems to be a more delicate test of infection than an agglutinative experiment (dilution 1 in 3, incubator temperature, limit 15 minutes); the agglutinative effect was never noticed until the index reached five times the normal, while after that (with one exception), it was always well marked.

3. The combination of this agglutinative effect and the opsonic determination will furnish in this disease a specific test of great value in diagnosis. The low opsonic power always found in normal or non-infected sera, so that even when moderately thick emulsions are used two or three cocci were the maximum found in any leucocyte, and the uniform absence of all agglutinative effect in all preparations containing normal serum forms a striking contrast with the very high opsonic effect found in preparations containing the serum from all cases of the disease on or after

the sixth day and in several cases before this date, such preparations always showing leucocytes packed with cocci and also dense clumps of cocci not yet ingested. *In fact, the picture is so characteristic that a diagnosis could in the majority of cases be made by simply mixing some washed corpuscles, serum from the patient, and meningococci, and incubating the mixture for a short time without the use of any control.*

4. From the very definite nature of the results obtained it would seem likely that this method will prove of value: (*a*) In diagnosing sporadic cases of the disease; (*b*) in settling the etiology of posterior basic meningitis in infants; and (*c*) in determining the nature of a doubtful coccus.

5. Two of the main elements on which the process of immunity in this disease depends are the opsonic and agglutinative powers of the serum. A therapeutic serum, therefore, having no opsonic or agglutinative power on the meningococcus could not be expected to have much value as a remedial agent in this disease. Several samples of serum on the market were tested and found to have neither opsonic nor agglutinative power. It may, however, be mentioned that the opsonic power disappears fairly rapidly and that a serum, active when freshly prepared, would soon lose its potency in this respect. From a few tests which were applied it would seem that this opsonin is very thermolabile and that a large thermal destruction even in the case of high opsonic sera derived from cases of the disease occurs by exposure to a temperature of 60° C. for 10 minutes.

H. H. Kutscher[1], who has studied agglutination in the meningococcus very thoroughly, finds that it is necessary in cultures seemingly of the meningococcus, in case of failure to agglutinate by a specific serum of high potency at 37° C., to attempt agglutination at 55° C. On the whole, however, strains agglutinating only with so high a temperature, are rare.

The opsonic index and agglutination in cerebrospinal meningitis have also been studied by Alice Taylor[2] in eight cases at the Children's Hospital, Great Ormond Street,

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(1) Deutsch. med. Woch., Nov. 15, 1906.
(2) Lancet, July 6, 1907.

London. The method of investigating the opsonic index was that described by A. E. Wright. The emulsion throughout was made from a culture on blood serum of meningococci isolated from the cerebrospinal fluid of one or other of the patients. These meningococci were all Gram-negative. The emulsion was used alive. The stain was a strong solution of toluidin blue made up with sodium bicarbonate; this gave satisfactory results. The leucocytes were washed once in a solution of sodium citrate in sodium chlorid and after that twice again in sodium chlorid 0.9 per cent. The emulsion was also made up with 0.9 per cent. sodium chlorid. Considerable difficulty was, however, encountered in dealing with the organisms, for it was found that the older cultures underwent rapid degeneration during incubation. In the later cases in which a seven to ten hours' culture was used this degeneration did not occur and in these cases a very high index has been noted.

While observing the opsonic index it was noted in the later cases that agglutination was a marked feature. This has been observed after an incubation of ten minutes in a dilution of 1 in 3. The controls throughout the whole series never showed any agglutination, nor was it present in slides without serum, where the emulsion was incubated with 1 part each of washed leucocytes and saline solution. It was also tested against tuberculous meningitis and typhoid fever; in neither case was agglutination present. In testing for agglutination alone a thick emulsion is to be preferred. The clumps are then very much larger and are spread along the edges of the smear in the same manner as the leucocytes, and the contrast between the control slide, thickly strewn with pairs and some tetrads of meningococci, and the patient's slide, almost or quite free from loose cocci but with clumps along the edges, is a striking feature.

From her observations, the author proposes and answers two questions:

1. Is a high opsonic index pathognomonic of the disease? It appears possible from these eight cases to obtain a definite opsonic index in cerebrospinal meningitis, either working with an old or a young culture. A high index has been shown to be present in all the cases examined. This index may under certain conditions attain a very high altitude. This high index appears to be due in these cases

to the characteristic feature that normal serum has scarcely any opsonic power against a young culture of this organism. It is therefore probable that a high opsonic index is pathognomonic of the disease.

2. Is a high opsonic index diagnostic in all forms of meningococcal meningitis? The results obtained on testing the serum of the present series of cases against the Belfast organisms show that a high index is not always obtained with different strains of growth and that a negative result may occur. So that although a high index indicates the presence of the disease. a normal index does not negative its presence.

Charles E. Simon[1] reports a case of cerebrospinal meningitis in an adult of 27, in whose blood the *meningococcus* was found in such numbers as to be *demonstrable in a blood smear.* The number of cases of meningococcus infection in which the organism has been found in the blood is as yet rather small. The leucocyte count was 50,000. Fully 8 per cent. of the leucocytes contained diplococci, which could not be distinguished morphologically from those found in the meningeal fluid of the same case, nor from diplococci isolated by culture from other cases of cerebrospinal meningitis. Like these, they were Gram-negative. It is noteworthy that the large mononuclear leucocytes were engaged in phagocytosis almost to the same extent as the neutrophiles.

The first case of meningococcus septicemia was reported by Gwyn in 1899 in the *Johns Hopkins Hospital Bulletin.* The same case was also reported by Osler.[2] A second case was reported by Salomon in the *Berlin. klin. Woch.* in 1902. Since then five other cases have been recorded. Including Simon's, there are thus only eight cases in which the meningococcus has been found in the blood. This is a remarkable fact considering the large number of cases of meningococcus meningitis which have been observed within the last few years; but it may find its explanation in the small number of blood examinations which have probably been made, as contrasted with examinations of the meningeal fluid in which the demonstration of the organism is no doubt more likely to lead to a definite result. Consider-

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(1) Jour. Am. Med. Assoc., June 8, 1907.
(2) Boston Med. and Surg. Jour.

ing, however that in the meningeal fluid even negative findings are not unusual, it seems advisable in all doubtful cases to resort to blood examination also. Simon views his own observation as a curiosity only and as not likely to be duplicated except on rare occasions. Nevertheless, a careful search should be made, which occasionally may lead to a fruitful result.

Symptomatology. C. L. Nedwill[1] reports a small epidemic of cerebrospinal meningitis in the Soudan. The epidemic occurred at the head of the railroad which was being constructed in the Nubian desert into the Dongola Province, and 22 natives were afflicted. The cause of the outbreak could not be traced, but it was suggested that the disturbance of the earth during railroad construction might have set the diplococci free. The epidemic occurred during June, July, and August, 1905. During June the shade temperature is usually about 112° F., often four or five degrees higher at midday, and at night falling to 70° or perhaps 60° F. The heat, over-exertion, and long hours of manual labor in the blazing sun were undoubted predisposing causes. Owing to the suspicious character of the natives, no trustworthy accounts of subjective symptoms could be obtained. Lumbar puncture was not done, there being at hand no needle long enough. Only one postmortem examination was possible because of the distrust shown by the natives.

The physical signs were the usual ones found in epidemic cerebrospinal meningitis. Thus there was delirium, stupor or coma, rigidity of the neck and spinal muscles, retraction of the head, vomiting, and restlessness. The temperature averaged 102° F. and the pulse 90. In no case was any form of rash detected, the dark skin of the native making the discovery of a slight rash rather difficult. Kernig's sign was present almost uniformly. Herpes labialis appeared in seven cases. Cranial nerve involvement was not uncommon. The mortality was high, there being 13 deaths in 22 cases.

Loss of knee-jerks throughout the illness proved to be a bad sign. "It would appear from a study of these 22 cases that non-recovery of knee-jerks within a week of the onset

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(1) Lancet, Dec. 1, 1906.

of the disease is an unfavorable sign." Details of the 22 cases are appended to Nedwill's report, and four accompanying temperature charts well illustrate the irregularity of temperature observed in these cases.

J. M. Mason[1] reports an epidemic of cerebrospinal meningitis occurring at Birmingham, Ala. Though of limited extent, the epidemic was one attended with a high rate of mortality, there being 78 deaths in 107 cases reported. Distributed according to race the statistics are: Whites, 34 cases, 26 deaths, mortality 76.4 per cent.; blacks, 73 cases, 52 deaths, mortality 71.2 per cent. Clinically the cases varied widely, some dying within a few hours of the onset, others surviving eight or ten days, others lingering for weeks and months, a few recovering promptly and some only after prolonged illness. The *Diplococcus intracellularis* was found in most cases studied bacteriologically, though tubercle bacilli were found in two cases, once in association with the diplococcus intracellularis; and the pneumococcus was found once. Twelve postmortem examinations were made in the series. Of these, the majority showed an acute purulent meningitis, extending over the surface of the hemispheres, but being particularly marked at the base. One patient died on the one hundred and second day from a chronic hydrocephalus, secondary to the cerebrospinal meningitis.

Mason is not of the opinion that the disease is very contagious. He regards it as only very mildly so. In only one instance did two cases occur in the same household. The onset in these two cases was within 36 hours of each other, infection of the two children doubtless being from a common source. Several other children in the same household escaped. Many of the cases were treated in open wards and failed to spread the disease.

The symptoms in the epidemic were the usual ones. Every case showed delirium and rigidity of neck and spine. Eye symptoms were uncommon, ptosis being noted in one case, strabismus in one case, and pupillary disturbances rarely. Eruptions showed in only three of twenty-one cases studied by Mason, herpes labialis appeared only twice.

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(1) New York Med: Jour., Nov. 10, 1906.

Rash in negroes is difficult to determine, and in a dark cabin it is not easy to observe accurately the pupils.

Lumbar puncture was performed in twenty of the twenty-one cases. The fluid withdrawn was cloudy in nine cases, opaque in two, purulent in five, and clear in three. In one case a fibrinous plug occluded the canula. Of the three cases with clear fluid one was tuberculous, one mixed tuberculous and meningococcic infection, and one meningococcic. Leucocyte counts, made in 13 cases, averaged 18,037, the extremes being 12,000 and 31,000. The temperature varied from 97° to 105° F. The pulse in many cases was as low as 48 or 50.

For the benefit of those readers who may desire to have a record of the technic of lumbar puncture, the author is quoted as follows: "The puncture is best made in the interval or between the third and fourth lumbar vertebræ. The third interspace corresponds to a line drawn across the back from the highest points of the iliac crests. A hollow aspirating needle of medium size, at least three inches long and preferably four inches for adults, is to be used. A wire trochar should be at hand, for it is sometimes necessary to pass it into the needle to remove blood clots, thick tenacious pus, or plugs of exudate. An aspirating syringe should never be used. The patient should be on his side with knees and thighs and trunk flexed, and with shoulders and head slightly raised on pillows. The needle should be introduced about one-half inch to one side of the middle line, and one-half inch to three-fourths of an inch below the tip of the spinous process of third lumbar vetebra. It must be pushed in an upward and inward direction.

"My aim in an adult is so to direct the needle that it will reach the middle line at a point about two and one-half or three inches deep. If the canal is not at once penetrated, the needle may be partially withdrawn and the point pushed in at a different place. Wherever bone is encountered it shows definitely that the needle has not reached the location of the interspace, and it must be searched for in another direction. In children and in thin individuals the canal is easily reached, but in stout adult persons it is frequently very difficult. Dry taps are sometimes made, i. e., the needle actually enters the canal, but no fluid is withdrawn. This may be due to thick exudate, stopping of canula with blood or tissue, and in case of secondary tap to adhesions around the point of aspiration. The fluid should be collected in sterile tubes and should be centrifugalized or allowed to settle, when the sediment should be spread on slides stained, and examined."

In the treatment it was found necessary to resort to morphin and bromids in practically all cases. Lumbar puncture is without marked curative effect, although it seems to give relief for a time. The hot bath treatment was comforting to the patient in every instance. Elevation of the head may possibly relieve cerebral congestion to a slight extent. Ergot was without effect. Diphtheritic antitoxin was not used, and no antiseptics were injected into the spinal canal.

G. A. Allan and J. S. Dunn[1] report a case of cerebrospinal meningitis of epidemic variety because of the comparative rarity of the disease in Scotland, and because of the fact that it was a fulminating case of the "convulsive comatose type" of Tourdes. To Americans, to whom epidemic cerebrospinal meningitis by reason of its frequent occurrence in the United States is well known, nothing noteworthy in the symptomatology appears. More interesting to them is the fact that this is the first case reported in Scotland, according to the authors, in which a bacteriologic examination was made after lumbar puncture, the very few cases previously reported in Scottish medical literature possibly being due to the pneuomcoccus of Fränkel or some other organism. A brief *résumé* of the Scottish cases shows the first case to have been reported in 1883, there being no authentic case in Scotland before that time. That same year a case was reported in which a micrococcus was found postmorten "among the pus cells" of the cerebrospinal fluid. In 1884 occurred the Galston epidemic of six cases, four being fatal. In 1885 a case was reported, 9 were reported in 1888, a case was reported in 1892, two in 1900. This is the entire list of known cases for Scotland. In an addendum the authors add two more cases to the one above reported.

That the rash in cerebrospinal meningitis is not well known in Scotland is evident from the description of four

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(1) Glasgow Med. Jour., September, 1906.

very typical cases reported by William Wright[1] from the Glasgow epidemic. In these cases the rash was purpuric, purple in color and hemorrhagic in character, in spots, with well-demarcated edges, varying in size from a pinhead to a bean. In one case in which death took place after 24 days' illness the rash appeared in the seventh day and came out in successive crops. The spots were definitely hemorrhagic, well calculated to justify the term "spotted fever."

Frederick Langmead[2] discusses the relationship between simple *posterior basic meningitis* and cerebrospinal fever. Attention was first called to posterior basic meningitis by Gee and Thomas Barlow in 1878. Lees in Clifford Allbutt's "System of Medicine," with Barlow, did much to establish it as a clinical entity. In 1898 Stiel found, as its cause, a diplococcus much resembling that found in epidemic cerebrospinal fever but differing from it in several respects. Langmead expresses the conviction [which we decidedly share.—EDS.] that the two diseases are identical, and gives a short *résumé* of the more salient points of the clinical and pathologic features of each disease, comparing them point by point. His conclusions follow:

1. Although there are certain clinical distinctions between posterior basic meningitis and cerebrospinal fever, yet by an appeal to the record of the former in older children those distinctions are lost.
2. Posterior basic meningitis can occur in epidemics.
3. The pathologic lesions of this disease have been too narrowly defined.
4. The slender bacteriologic distinctions are being nullified by recent experiment.
5. The diplococcus, as described by Still, has been isolated from the blood of a case of malignant epidemic cerebrospinal fever.

Treatment. *Medical.* W. O. Wilkes[3] reports a case of cerebrospinal meningitis in a boy of 10 years, the illness keeping him in bed seventy days. As to symptoms the case was a usual one and need not be summarized. Wilkes came to the conclusion that in the treatment of this condi-

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(1) Lancet, Sept. 15, 1906.
(2) Practitioner, April, 1907.
(3) Med. Record, Nov. 17, 1906.

tion iodid, bromid, bichlorid or gelsemium are without any beneficial effect whatever, if they are not actually harmful. Ergot may have a limited usefulness, chiefly to aid the quieting effect of morphin. Blistering is worse than useless and adds to the pain and suffering, if not to the weakness of the patient.

The chief remedies are morphin hypodermically and ice-bags to the head and spine. Possibly small doses of chloral by the bowel, or internally, will help, if the morphin does not produce sleep. Spinal puncture is beneficial in that it relieves pressure symptoms. Albumin water, solutions of peptones and milk should be given for food. Strychnin and alcohol are advisable if the heart needs support.

Wilkes is of the opinion that the disease is but very mildly contagious since the boy was shut up in a house for ten weeks with four adults and five children, without any other case developing. He is further of the opinion that it is folly to quarantine cerebrospinal meningitis and to disregard pneumonia, which is just as contagious.

Mechanical. Henry Koplik[1] discusses the application of percussion of the skull as a means of determining the indication for the performance of *lumbar puncture* for therapeutic purposes. As a therapeutic measure lumbar puncture has been applied in hydrocephalus, acute and chronic, whether congenital or secondary to meningitis, and also in cerebrospinal meningitis, chiefly for the purpose of removing an excess of fluid present.

There is no monograph extant which will give a student any idea as to when the proper moment arises in cerebrospinal meningitis, either to apply lumbar puncture for the first time, or when to repeat it. We are repeatedly told that lumbar puncture is performed to relieve pressure on the structures at the base of the brain and subarachnoid space, but there is no work which will tell the physician how to diagnose intracranial pressure, as actually present, before proceeding to puncture. There may be delirium, excitement, hyperesthesia or headache present in a case of meningitis as a result of the toxemia without the presence of intracranial pressure.

Given a case of meningitis, the first result of intracranial

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(1) Med. Record, Sept. 29, 1906.

pressure is an acute, subacute, or chronic distension of the ventricles of the brain with fluid, and it is the early detection of this fluid which gives us an absolute indication to relieve distension by lumbar puncture. The mode of detecting acute hydrocephalus, whether caused by acute distension of the ventricles on the first day of a cerebrospinal meningitis, or during the slow, insidious onset of a tuberculous meningitis, is by percussion of the skull. McEwen, in his work on the pyogenic diseases of the brain and spinal cord, describes very accurately the elicitation of a differential cranial percussion note, in which he shows that in various conditions, such as cerebral tumors, the lateral ventricles are distended with serous fluid. In such cases the percussion note elicited at a given spot on the cranium, such as the pterion, varies according to the position of the head. While the person sits with the head upright, the most resonant note is brought out by percussion of the skull by means of the finger as in chest percussion towards the basal level of the frontal bone, and the squamous portion of the parietal. The percussion on the lower bone, as the head is inclined to one side, gives a tympanitic note. As McEwen says, the exact musical quality of the note is difficult to describe, but when heard gives an idea of hollowness. McEwen found this note in over forty children and young adolescents, who had distended ventricles due to various causes.

In no work do we find any data as to the application of percussion of the skull in cases of cerebrospinal meningitis. Koplik considers percussion of the skull the only definite means we have today of determining whether there is an increase of fluid in the ventricles or the subarachnoid space causing pressure effects, and therefore demanding immediate relief. Moreover, percussion of the skull is a useful mode of differentiating cases of so-called meningism from true cases of meningitis. Koplik reports in this connection a case of pneumonia with cerebral symptoms denoting meningitis, and leading the attending physician to propose lumbar puncture for the purpose of relieving symptoms; percussion of the skull showed no evidence of an accumulation of fluid in the ventricles, and a lumbar puncture was not made. The after course proved that meningitis had been merely simulated, and was never really present.

How are we to know whether repeated puncture is necessary in a given case? If, after puncture, the headache disappears and the patient becomes rational, even though pulse and fever run high, delay is proper. If, however, the patient's symptoms remain, after puncture, quite uninfluenced, it will be found that the percussion sign of hydrocephalus continues, and the second puncture may be made in from twenty-four to forty-eight hours.

Vaccine Therapy. Experimental cerebrospinal meningitis and its serum treatment is discussed by Simon Flexner[1], his work being a contribution from the Rockefeller Institute for Medical Research, based on study into the epidemic which prevailed in New York during the winter of 1904 and the spring of 1905. There were in this epidemic about 4,000 cases, of which 3,429 died, a mortality of 73.5 per cent. Children were the chief sufferers, for the most part living in the crowded tenements of the congested districts. Flexner first reports his studies on the diplococcus itself, and then recounts his experiments on guinea pigs and monkeys, making free use of lumbar puncture in the latter animals.

The antiserums made in large monkeys (*Macacus nemestrinus*) were used on a series of five smaller monkeys (*Macacus rhesus*), which had been injected with fatal quantities of the diplococcus. The control monkeys of this series all died within twenty-four hours. The first monkey received the serum and suspension of cocci simultaneously into the spinal canal; it remained well. Lumbar punctures showed a moderate reaction and rapid disappearance of the cocci, partly by phagocytosis but chiefly, apparently, by lysis within the spinal canal. The second monkey was given the serum two hours after the injection of cocci; it became very sick, but recovered. The exudation was much more marked in this animal, and the cocci were only in part taken up by leucocytes. The third monkey was injected with serum five hours after the cocci were given. The disease, which had already manifested itself, was arrested. The next day the animal appeared well. Lumbar puncture gave a fluid showing many leucocytes with an occasional intracellular coccus, and many extracellular cocci. These

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(1) **British Med. Jour., Oct. 20, 1906.**

three monkeys were treated successfully with the serum by *intraspinal* injection. The next monkey was treated successfully by a *subcutaneous* injection of 5 c. c. of the serum. The injection of cocci was made at 8:45 a. m., and of serum at 11 a. m. The animal, which was already depressed before the injection of serum, remained in about the same condition for a few hours and then gradually improved. The next day it appeared to be well.

This series of experiments was, up to this point, successful. But he records a failure under conditions which, in view of the foregoing tests, was wholly unexpected. The monkey was injected with the serum intraspinally three hours after having received the emulsion of cocci. The already sick monkey grew progressively worse and died nineteen hours after infection. The autopsy showed an unusually heavy exudate in the membranes of the cord, and smear preparations showed the exudate to be purulent and the cocci all to be within leucocytes. The cocci were recovered in cultures.

In how far the results obtained with guinea-pigs and monkeys can be applied to the prevention and treatment of cerebrospinal meningitis in man it is not safe to predict. Should further experiments show that subcutaneous and intravenous injections of the antiserums can modify favorably the experimental disease in monkeys, the case would not be without hope. Even the injection of immune serums into the spinal canal in man might be thought of as an eventual mode of combating the disease, once the serum is proven to be really effective in monkeys and the injection of an alien serum to be without prejudicial effect.

A case of cerebrospinal meningitis treated by vaccine injections with recovery is reported by H. Mackenzie[1]. The case was the first to be reported in London during the recent epidemic. "Although it was a very severe type of the disease, at first suggesting the malignant form, it happily ended in recovery. No conclusions of any value can be drawn as to the effects of treatment in one case. With regard to lumbar puncture, after an extended use of this method of treatment, he considers it of great value in all forms of meningitis. It should be performed as soon as

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(1) British Med. Jour., June 15, 1907.

one diagnoses the nature of the case, and should be repeated at short intervals, especially where there are signs of intracranial pressure. The immediate improvement in the patient's condition after lumbar puncture is usually most striking. The fluid should be allowed to come away gradually drop by drop, as it usually does, and should be collected in a test tube.

"In this case in nine punctures nearly 9 oz. of fluid were removed. In two punctures no fluid was obtained. The fluid obtained was at first very turbid, then was clear, again was very turbid on the fourth puncture, while afterwards it was either opalescent or clear. The cells contained in the fluid were throughout chiefly polymorphonuclears. The micro-organisms were intracellular, Gram-negative diplococci, and on one occasion the *Staphylococcus albus.* After the end of the fifth week the fluid was sterile.

"The vaccine used was prepared and administered by Leonard Dudgeon. It is obvious, however, that the value of such a vaccine can only be determined by experience in a sufficient number of cases. It is impossible to say how far the recovery was due to the lumbar punctures and the vaccine injections respectively."

C. Rundle, J. C. Mottram, and R. S. Williams[1] report a case of cerebrospinal meningitis, also with isolation of the specific organism, the preparation of a vaccine, its administration, and recovery, in a child only six months old. From the time of the infant's admission until the commencement of the vaccine treatment his condition became steadily worse. Spasm of the muscles of deglutition was induced by each attempt to swallow, and the extreme retraction of the head and neck appeared to offer a physical obstruction to the passage of food.

The first vaccine inoculation was made March 11. With the exception of a remission in the temperature of the body no marked change was observed in the clinical manifestations during the succeeding 48 hours. On March 14, however, a definite decrease in the degree of extension of the head and neck was noted, the spine could be straightened with the exercise of moderate force, and lateral movements

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(1) Lancet, July 27, 1907.

of the head were made voluntarily. A second inoculation was made March 16, followed by a period of apyrexia lasting 24 hours. On March 17 the eyeballs were observed to be fixed less rigidly, some lateral movements occurred voluntarily. The improved position of the head and neck at this period made feeding by the mouth again possible. Vomiting ceased entirely. Hyperesthesia and Kernig's sign persisted. During the period from March 17 to 21 the temperature was raised and irregular; the third inoculation was made on the latter date, followed, as were the previous inoculations, by a period of apyrexia persisting on this occasion six days. The squint was now occasional only, the position of the head and neck approaching the normal, and the flexion of the joints of the extremities was easily overcome. A moderate degree of pyrexia from March 27 to 29 was met by a further dose of vaccine on the latter day. The temperature subsequently remained normal until the end of treatment. Kernig's sign was first noted as absent on April 2. From this date to the conclusion of treatment a rapid and progressive improvement in the condition of the child was maintained. No affection of the special senses persisted.

Preparation of Vaccine.—A 24-hours old culture was inoculated into sterile physiologic saline solution and placed in a water bath of 60° C. for half an hour; nasgar tubes were inoculated from it and incubated overnight to test sterility. The number of cocci contained was estimated by mixing known volumes of washed normal blood and vaccine; these were then diluted with physiologic saline solution, films prepared at once and stained by Leishman's method. The number of organisms present was counted against the red blood corpuscles. The vaccine was freshly prepared for each of the first four doses; the fifth had been made seven days before use.

Opsonic Index.—Equal parts of leucocytes, emulsion (24-hours old culture in 0.1 per cent. NaCl), and serum were mixed together and incubated at 37° C. for ten minutes; it was found that incubation for 15 minutes made the count extremely difficult to perform from excessive vacuolation and rapid degeneration of the cocci. The smears were stained by Leishman's method; at least 200 leucocytes were counted. The first inoculation (200,000) was made March 11, the index having been then 0.7 on two consecutive days; on the twelfth it rose to 1.5 but fell again to 0.9 and 1 on March 13, 14, 15 and 16. As this dose had produced no harmful effects, but had been followed by temporary alleviation, it was repeated (500,000) March 16. A further dose (510,000) was given and a slight fall on the twenty-second was followed by an abrupt rise on the twenty-third. Within 48 hours the index had again dropped markedly. The events following the first inoculation had repeated themselves. As the index remained comparatively low on March 25, 26, 27 and 28, with a slight tendency to rise of temperature on the last two days, a fresh dose (530,000) was given on the twenty-ninth. The index fell slightly, followed by a rise which was well maintained until April 4. On the fifth the index had dropped to 1.1. What proved to be the last inoculation was made on this day. A slight rise was noted on the 8th. Since the inoculations were on each occasion followed by a rise in the index, and on three occasions by a rise of so marked a character, it seems reasonable to suppose that there is a connection between the two events.

Other Forms of Cerebrospinal Meningitis. *Rheumatic.* Lépine[1] reports a case of spinal meningitis of rheumatic origin, associated with root symptoms and occurring in a patient with chronic articular rheumatism. Interest does not center in the spinal meningitis of rheumatic origin, since well authenticated cases have been observed by Teissier, Fouche, Léri, Triboulet and Thomas, Klippel, and others; but rather in the fact that it is in this case associated with chronic rheumatism instead of the acute variety. All meningeal reactions, whether slight or severe, are the result of invasion of the cerebrospinal fluid by poisons, bacterial or metabolic. The irritation increases the amount of cerebrospinal fluid, its tension rises, and pressure symptoms, chiefly pain, follow. Then the pia mater becomes inflamed.

The patient described by Lépine was a man of 50 years of age, giving neither a syphilitic nor tuberculous history. The onset was with symptoms suggesting lumbago, but associated with hyperesthesia in the territory involved,

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(1) Lyon Méd., 1906, p. 342.

slight atrophy, and Kernig's sign. Lumbar puncture gave issue to a jet of cerebrospinal fluid, which was evidently under great pressure. The fluid was free from bacteria, poor in cells, without either lymphocytes or polymorphonuclear cells. It contained, however, a considerable quantity of fibrin, a fact which well comports with the rheumatic nature of the case. If a lymphocytosis had been found syphilis must have been expected.

Streptococcic.—E. E. Southard and R. R. Stratton[1] have made a study of acute leptomeningitis caused by the streptococcus pyogenes, first as to the age of the exudations, secondly as to certain prominent vascular lesions, and, thirdly, as to the neuroglia reaction. The study is based on nine cases, five being due to otitis media. Seven patients were male, two were female. Seven patients were over 30 years of age, and two of these were over 50 years. One was an infant, and one was 19 years old. The brain lesions found varied in character and distribution. The leptomeninges showed lesions in all cases.

The distribution of the pial exudate was interesting. Except the hydrocephalic, the base showed an exudate in all cases. The lesions of the brain substance were various. Flattening of the gyri stands in close relation with involvement and probably with actual distension of the ventricles. When flattening is absent, the substance is soft and moist, and may be finely injected. No case of cortical flattening fails to show exudate in the ventricles; but in early stages of ventricular exudation the sulci may flare normally.

Clinically viewed, the cases are of various duration and run a trifle longer than the pneumococcus cases. The relation of streptococcus meningitis to lung lesions is less suspicious than that of staphylococcus or pneumococcus brain infections to lung lesions. The part played by lung lesions in bringing about meningitis has probably been overestimated. The streptococcus cases seem to be more pronouncedly "cerebral" or "meningitic" from the very start of acute symptoms than are the pneumococcus and staphylococcus cases, which are a little more "primary."

Anatomically viewed, the streptococcus cases preserve their resemblance to the pneumococcus cases, even to the

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(1) Jour. Am. Med. Assoc., Oct. 20, 1906.

production in some cases of small abscesses. Considered histologically, the streptococcus cases again resemble the pneumococcus cases. The same relations of bacteria to leucocytes, of leucocytes to cells of the macrophage type are shown in the two series. The intimal infiltration of the arteries and the intimal proliferation and exudative lesions of the veins run parallel in the two cases. The occurrence of early fibrillar gliosis (in the sense of the production of new fibrils by the swollen neuroglia cells) is important.

Bacillary.—A case of purulent cerebrospinal meningitis is reported by C. F. Noeggerath[1], in which the *Bacillus coli immobilis capsulatus* (Wilde) was found. The patient was an infant 16 days old, well until the eleventh day, when convulsions appeared. There was retraction of the head, without marked rigidity or opisthotonos. Cheyne-Stokes breathing was observed. Reflexes were everywhere increased, but the Babinski reflex was lacking. [A Babinski reflex in a child under one year of age has no diagnostic value.—ED.]. Without going into the complete symptomatology, it is enough to say that the author entertained the possibilities of cerebral hemorrhage, navel sepsis, and purulent cerebrospinal meningitis. Lumbar puncture was made and only a very few drops of purulent fluid which blocked the canula, were obtained. This exudate showed microscopically a bacillus identified by bouillon, agar, gelatine and potato cultures as the *Bacillus coli immobilis capsulatus.* This diagnosis was confirmed in its entirety as to disease and cause by the postmortem examination. The type of cerebrospinal meningitis caused by this germ in no way differed from that caused by the diplococcus.

Tuberculous.—Georg. Riebold[2] reports a case of, presumably, tuberculous cerebrospinal meningitis, resulting in recovery, and based on his experience with this case, he advises that daily spinal punctures be made in such cases. Still, he admits that three other cases of tuberculous cerebrospinal meningitis so treated died.

Influenzal.—A case of influenzal cerebrospinal meningitis is reported by J. Sholto C. Douglas,[3] the patient being a 10-months-old female. The symptoms may be here omit-

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(1) Münch. med. Woch., March 26, 1907.
(2) Münch. med. Woch., Aug. 28, 1906.
(3) Lancet, Jan. 12, 1907.

ted since they are the usual ones of cerebrospinal fever. A postmortem examination was made about twelve hours after death. The brain was well developed but was much swollen and somewhat softened and its convolutions were flattened. Over its surface was a quantity of thick green pus, especially in the frontal region, anterior to the fissure of Rolando; and the fissure of Sylvius was occluded by pus. Slight congestion of the brain substance was evident in section. The pus did not penetrate deeply into the fissures, but the fornix and corpus callosum were softened and infiltrated with pus. Examination of the cerebrospinal fluid obtained during life by lumbar puncture showed the influenzal bacillus of Pfeiffer, and no tubercle bacilli. Douglas affirms that his is the first case of influenzal meningitis reported in England, in which the organism was obtained both from culture of the cerebrospinal fluid during life and from the meningeal exudate after death.

Cortical Localization.

Progress of Investigations. During the past year a great many articles have appeared on the general subject of localization in the cortex. While agreement in general reigns at the present time regarding localization in the motor cortex, most of the remaining cortex is still a matter of argument. Some very iconoclastic articles have appeared during the year, stimulated by two from the pen of Pierre Marie, tending to spread disbelief in the existence of the time-honored motor speech center in the third left inferior frontal gyrus (Broca's convolution) in right-handed people. Some of these articles are summarized briefly.

Prefrontal lobes. The function of the prefrontal lobes is discussed by Alfred Gordon[1], based on the pathologic findings in a case which he reports. Of the cases in the literature it may be said that sifting all the accumulated data the conclusion can be drawn that a tumor or any lesion of the prefrontal region, particularly of the left, is more frequently attended by mental disturbances than a lesion of any other portion, except, perhaps, of the corpus callosum. The following are the principal symptoms: Mental hebetude, automatism, excitement and irritability,

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(1) Jour. Am. Med. Assoc., April 27, 1907.

or else depression, disorientation and loss of power to concentrate attention. The German writers long ago called attention to a special symptom, which they found in a large number of cases in diseases of the prefrontal lobe. Jastrowitch gave it the name *Moria;* Oppenheim calls it *Witzelsucht.* The characteristic feature of this symptom is the humoristic spirit which is seen in the patient's actions and words.

The patient whose history is related presented during life, in addition to excitability, disorientation, motor restlessness, indifference to surroundings, inability to concentrate attention, all symptoms to some extent characteristic of prefrontal lesions—a state which can be called "euphoria." At autopsy a recent hemorrhage was found in the left prefrontal lobe.

This case is interesting because of the causal relations of a lesion in the left prefrontal region to the mental phenomena and because of the special character of the latter. The euphoria of the patient was striking; he expressed a psychic childishness in his acts and words. Since it was the most conspicuous phenomenon throughout the man's brief illness, perhaps it could be considered to some extent as pathognomonic of diseases of the frontal lobe.

Frontal Lobes. S. J. Franz[1] reports experiments on monkeys made with a view of studying the functions of the association areas of the cerebral cortex. He succeeded in teaching monkeys to perform certain acts demanding an association of ideas, and then the frontal lobes were cut away, the animals allowed to recover, and tests were made of their memory for these associations. The author's conclusions follow:

1. In monkeys, as well as in cats, the frontal lobes are normally employed in the formation of simple sensory associations.
2. When the frontal lobes are destroyed, recently formed habits are lost. It has been found possible, however, for the animal to form new associations or to relearn old tricks.
3. When the associations are firmly established, destruction of the frontals is not always followed by a loss.

There are all degrees of "memory" for any such particular habit, from perfect to a very decided hesitancy and slowing. The experiments show not only that there are mental changes coincident with disturbances of the frontal lobes, but that the mental change is one that could not be determined by simple observational methods, and they suggest, furthermore, that, for the determination of corresponding changes in man, accurate psychologic or psychophysic methods should be employed.

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[1] Jour. Am. Med. Assoc., Nov. 3, 1906.

Motor Cortex. Hermon C. Gordinier[1] reports three cases of disease of the motor area which are of interest as tending to prove that this area lies in front of the fissure of Rolando.

CASE I was that of a glioma destroying the upper third of the precentral gyrus and its paracentral annex, with the bases of the first and second frontal gyri, which at first excited crural spasm and monoplegia, followed by paresis of the arm and face, the latter symptoms being probably due to subsequent softening of the remainder of the precentral gyrus.

CASE II was that of an abscess of the middle of the right precentral gyrus and bases of the superior and middle frontal gyri, which caused unilateral convulsive movements of the head and face toward the left, together with similar movements of the left arm and face, followed by complete motor paralysis of the left arm and paresis of the lower facial muscles of the left side.

CASE III presented a broken-down gumma with the formation of scar-tissue and consequent atrophy of the cortex of the lower part of the left precentral gyrus, and caused, for a period of about three years, right unilateral facial spasm, followed by paresis of the face without motor aphasia; death probably was due to uremia.

All three cases presented during life focal convulsive movements followed by partial or complete paralysis of motion, not preceded by auræ, and the most careful examination failed at any time to disclose the presence of sensory symptoms. The causes, all verified at autopsy, were lesions situated anterior to the fissure of Rolando, in the precentral gyrus, together with the bases of the superior and middle frontal gyri.

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(1) Am. Jour. Med. Sciences, May, 1907.

In a study of 22 cases collected from the literature, either with autopsies or with exposure of the cortical area at operations, and which presented during life motor paralysis, preceded or not by Jacksonian epilepsy and without sensory symptoms, the lesions were invariably located anterior to the fissure of Rolando, involving the precentral convolution together with, in several instances, the bases of one or more frontal gyri.

Of great value in support of the doctrine of the definite localization of motor and sensory centers distinct from one another are the cases of partial or complete sensory loss without motor symptoms, due to lesions located dorsal to the fissure of Rolando in the parietal lobe. Redlich, from a study of the position of the lesions in 20 cases, with defects or loss of the muscle sense, believes that we can positively locate a center for muscle sense in the parietal lobe. The author draws the following conclusions:

1. There is a determined area in the cerebral cortex presiding over motor functions, and that area is the precentral convolution and its paracentral annex (possibly the bases of the superior and middle frontal gyri may have to be included).

2. The motor and sensory areas of the brain, though interdependent, are, as in the spinal cord, perfectly distinct from one another.

James H. Lloyd[1] discusses the findings obtained from faradization of the motor cortex in the human brain. There are some details of technic which must be observed. In human subjects a large osteoplastic flap should be made. This exposes the patient to no increased risk, gives the surgeon a better opportunity, leaves a better permanent result, and permits of more ample faradization. The unipolar method should be used, as this allows of a concentrated effect not possible when both poles are used. Observations were made by Lloyd on two cases, one of which is reported in detail. He concludes that "so far as observations on one case can determine the location of the motor centers entirely in front of the central fissure, the present observation is entirely in accord with this claim. The reactions were prompt when the precentral gyrus was fara-

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(1) Jour. Am. Med. Assoc., Dec. 15, 1906.

dized, and there was no response when the postcentral gyrus was touched." Thus again the conclusions of Sherrington and Grünbaum, as to the location of the motor cortex, are confirmed.

An *abnormal response* to irritation of the motor cortex is reported by Alfred Gordon[1], in a patient with left-sided Jacksonian epilepsy. The test was made by the unipolar method during operation. The blunt end of a very small metallic electrode covered with a thin layer of absorbent cotton was lightly applied to the exposed *right* motor cortex. In accordance with the suggestion of Sherrington and Grünbaum, application was made first to the ascending parietal, and lastly to the ascending frontal gyrus. Every time the electrode touched the brain tissue contractions would be noticed on the same side of the body (*right* side). Application to the upper angle of the exposed area would produce distinct contractions in the arm and very slight in the leg; when the electrode was applied to the middle area, the arm alone would twitch and faradization of the lower angle brought on contractions of the arm, neck, and face. The remarkable phenomenon was the fact that at no moment convulsions or any twitching was noticeable on the left side. While the response was decidedly marked upon irritation of the ascending frontal, it was much less pronounced when the ascending parietal was faradized. Nevertheless, irritation of the latter gave some twitching of the extremities. The striking feature is the presence of homolateral muscular contractions. The only explanation, in the author's judgment, is an anomaly of the pyramidal tract.

Parietal Lobe. Charles W. Burr[2] calls attention to *tactile amnesia* as a diagnostic localizing sign. Mere tactile anesthesia does not lead to inability to recognize objects held in the hand; but when anesthesia affects the underlying tissues so that deep pressure is not felt, the patient not only does not know what he has in his hand, but does not know that he has anything. More important than light touch is the ability to know the position of the fingers, to differentiate weights, to locate the point touched, and to recognize the number of points of contact. Failure in these

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(1) Med. Record, June 22, 1907.
(2) Am. Jour. Med. Sciences, July, 1907.

regards, even if mere touch sensibility is preserved, leads to true astereognosis. This is not to be confused with tactile amnesia. In the latter sensibility to all forms of stimuli is preserved, but the patient, though knowing he has something in the hand, cannot recognize what it is. It is not that he cannot recall its name (tactile aphasia), but that he does not know its nature or uses, or anything about it. [Sometimes called tactile apraxia.—ED.] The recognition of an object is a psychic process, and somewhere in the cortex is an area where memories of passed tactile sensations are stored up. Disease of this part of the cortex or its association pathways would lead to tactile amnesia.

"The location of this center probably is in either the posterior part of the parietal lobe or the anterior part of the convex surface of the occipital lobe. The frequency of the association of the symptom with word blindness and mind blindness makes it probable that the center is near the higher visual center. It cannot be identical with the center for common sensation, because tactile sensibility is not affected. The frequent occurrence of the symptom with mixed sensory aphasia due to a unilateral lesion would indicate that the center is *unilateral*. It must be posterior to the fissure of Rolando. The symptom never occurs if the area of disease involves only the prefrontal lobe. I have never seen it in pure motor aphasia, in uncomplicated left-sided hemiplegia, nor in right-sided hemiplegia, unless there is also a greater or less degree of word blindness or object blindness or some type of sensory aphasia. There may, however, be complete mind blindness, an absolute inability to recognize any object by sight, without any tactile amnesia."

In differentiating it from astereognosis it must be remembered that there is no disturbance of primary sensibility and that both sides are affected. Its presence or absence cannot be determined in severe cases of total or of mixed sensory aphasia, because it is impossible to make such patients understand what they are to do when an object is put in the hand. The only patients suitable for examination are those in whom the aphasia is partial or in whom recovery has gone far enough for the patients to understand short and simple commands and to use language enough to show whether they know what they are grasping.

Burr reports seven cases in support of his ideas, but in only one was a postmortem examination made. The patient in life showed word blindness and partial tactile amnesia coming on after a sudden hemiplegia and followed by complete dementia, caused by arterial sclerosis and a brain tumor. The latter, at the autopsy, was found infiltrating the whole left parietal lobe and the anterior part of the occipital lobe. It did not pass the fissure of Rolando anteriorly. Yet even in this case Burr confesses that "the lesions were so widespread that the case could not be used to locate any function."

Conjugate Deviation of Eyes and Head. Conjugate deviation of the eyes and head, and disorders of associated ocular movements in tumors and other lesions of the cerebrum, are thoroughly discussed by T. H. Weisenburg[1]. Furthermore, he reports 16 relevant cases. Experimental investigation on lower animals shows that electrical excitation of any portion of the brain, if of sufficient intensity, is productive of deviation of the eyes, or of the head, or both. There is good reason for this. Perhaps no function is more highly specialized than that which is concerned with the movements of the head and of the eyes. Such specialization of function necessarily needs and acquires a most complex cortical representation. Summing up the views so far advanced, Weisenburg is of the opinion that the movements of the head and eyes are dependent on a most complex mechanism; that there is only one true or at least functionally important cortical motor center anterior to the central fissure; that this motor center is in immediate connection with the sensory portions of the brain; that whenever the head and eyes are moved it is the result of a sensory stimulation, and that any interference with the sensory-motor arc will cause impairment of this function.

In the 16 cases with necropsy reported in his paper, lesions were found in almost every portion of the brain. If we consider more minutely the location of the lesions causing conjugate deviation, we find three principal areas involved: 1. The frontal area. 2. The area of the angular gyrus. 3. The internal capsule. It is the opinion of

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(1) Jour. Am. Med. Assoc., March 23 and 30, 1907.

some French writers, notably Bard, that to cause deviation there must always be an involvement of the optic radiations. This, however, is not correct, as in most of the recorded cases, including those described by Weisenburg, there was no such involvement.

From the pathologic evidence the conclusion must be drawn that lesions involving the posterior portions of the second and third frontal convolutions and the adjacent precentral convolution cause conjugate deviation of the eyes and head, and that when lesions elsewhere in the brain produce conjugate deviation there is an interruption either of the projection fibers in or near the internal capsule or of the association fibers between the occipital and temporal cortex and the frontal lobe. The following conclusions can be drawn as a result of the studies in Weisenburg's paper:

1. Conjugate deviation of the eyes and of the head is dependent on a most complex mechanism.
2. In the human being there is but one oculomotor center, or at least one active functionating center, situated in the posterior portion of the second and third frontal convolutions, adjacent to the precentral convolutions.
3. A separate center exists for the movements of the head, probably in the lower anterior portion of the precentral convolution.
4. There is probably in man a distinct center for the combined movements of the eyes and head, situated in the area between the head and the eye centers.
5. It is probable that the cortical oculomotor, head and combined head and eye centers are subdivided for lateral as well as for upward and downward movements.
6. The oculomotor and the motor head centers are in connection by means of association fibers with the cortical centers for the special senses, in the temporal, occipital, uncinate and other lobes.
7. Any lesion in the motor centers for the eyes and head or in the related special sense centers or in the association fibers connecting the former with the latter will cause an impairment in voluntary deviation of the eyes or of the head, or of both, this depending on the nature and location of the lesion.
8. Lesions in the angular gyrus cause conjugate devia-

tion because of involvement of visual and auditory fibers which lie underneath this area.

9. The theory of Bard that conjugate deviation of the eyes and head is always or nearly always accompanied by homonymous lateral hemianopsia and dependent on this, is an error, for in the majority of instances hemianopsia does not exist.

10. Hemianopsia may be caused by the shock or transient effects of the hemorrhage, this loss of half vision being only a temporary symptom and similar in nature to the temporary hemianesthesias sometimes observed in capsular lesions.

11. *The occurrence of conjugate deviation of the eyes or of the head, or of both, is of no value as a localizing symptom, because it may be the result of a lesion in any portion of the cerebrum. It may be of value, however, in conjunction with other localizing symptoms.*

12. Conjugate deviation of the eyes and head probably occurs in every case of large apoplectic lesions, but in some instances it is a partial or minor form and of transient duration.

13. Paralysis or impairment of associated ocular movement may occur as a result of a hemianopsia. This, however, is only temporary.

14. There is at present no evidence of a center or centers for automatic ocular movements, but if such centers exist in the thalamus a lesion of these should cause forced or incoördinate ocular movements.

15. Convergence and divergence are probably not reflex acts, but associated ocular movements similar to lateral and upward movements. They probably have cortical centers in the posterior portion of the second and third frontal convolutions. The movements of convergence and divergence are probably brought about by associating tracts in the pons and cerebral peduncles.

In the discussion following the reading of Weisenburg's paper, Charles K. Mills stated that he did not believe that the only cortical ocular center is situated in the posterior portions of the second and third frontal convolutions. That the only actively functioning center in the whole human being is situated in this region can be accepted as a practical fact based on clinical and pathological observations.

Dr. Mills believes that there is a motor region in lower animals and in man for every cortical region of the special senses. For practical purposes of diagnosis, however, the only one that claims attention in the human being is the musculocutaneous or main motor region. The human being has an auditory motor region, a visuo-motor region, etc. As shown by the investigations of Flechsig and the experiments of Grünbaum and Sherrington, Schäfer and others, motor centers are situated along the calcarine and parieto-occipital fissures and the posterior extremity of the intraparietal fissure. These centers may be largely in abeyance in the human being, their place having been taken as the result of racial and individual evolution by the more frequently employed oculomotor centers of the main motor area.

W. G. Spiller regards as the most important conclusion of Weisenburg's, that which states that conjugate deviation of the head and eyes cannot be used as a clinical sign of lesions of any particular part of the brain.

APHASIA.

During the past year there has been much controversy over the subject of aphasia, Pierre Marie having declared that there is no evidence that there are such things as sensory and motor aphasia, but that aphasia is a unit, and is merely a variety of intellectual deficit. In support of his proposition he attempts to show (and he has succeeded in showing), that the third inferior frontal convolution, known as Broca's convolution, is not the center for motor speech. Space forbids the summary of all the articles which have appeared on this general subject.

[For those having access to the *Neurologisches Centralblatt* a whole series of summaries of important articles can be found in the issue for Aug. 1, 1907, pages 709-722. Marie's long articles are to be found in *La Semaine Médicale,* Oct. 17 and Nov. 28, 1906, and an echo of Marie's first paper comes from the pen or F. Bernheim, in *La Semaine Médicale* for Nov. 7, 1906.—ED.]

The best *résumé* of the present controversy appears in an article by F. H. Dercum[1]. He discusses Marie's views

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(1) New York Med. Jour., Jan. 5, 1907.

on aphasia, first calling attention to the very slender basis on which rests our belief in the rôle played by the left third frontal convolution in motor aphasia. Fränkel and Onuf[1] collected 104 cases of motor aphasia; seven of these showed a lesion limited to Broca's convolution. In five of these seven cases the aphasia was merely transient or temporary; in only two was it persistent. "Marie's utterances on the subject of aphasia are both remarkable and revolutionary. Briefly stated he holds that the chief characteristic of aphasia is an intellectual deficit; that aphasia is a unit, that it is not made up of sensory aphasia on the one hand, or motor aphasia on the other, but that by lesion of the zone of Wernicke, there is established an intellectual deficit for the comprehension of spoken language; that in so-called sensory aphasia or aphasia of Wernicke, the lesion involves the zone of Wernicke (i. e., the supramarginal gyrus, the *pli courbe*, and the posterior portions of the two first temporal convolutions); that in so-called motor aphasia there is in addition to a lesion of this zone of Wernicke, also an involvement of the region of the lenticular nucleus. Lesion of the lenticular nucleus gives rise to anarthria; therefore in so-called motor aphasia, or Broca's aphasia, we have merely ordinary Wernicke's aphasia *plus* anarthria. Marie denies that the third left frontal convolution plays any rôle in aphasia whatever. He believes that the involvement of this convolution seen in so many cases—in half of Marie's cases—is purely a coincidence, due to the extent of the vascular territory obliterated and to nothing more."

Dercum examined recently fourteen cases of aphasia, and, in addition, three cases of pseudobulbar palsy, one of which came to autopsy. Attempts to classify them as sensory and motor failed, since all presented sensory elements. They could be roughly divided merely into a group in which motor speech disturbances were more pronounced, and a group in which they were less pronounced. Moreover, as Marie insists, all these aphasics showed an intellectual deficit, some being unable to comprehend a single word, and others being able to comprehend only simple sentences, written or spoken.

Of the fourteen aphasics seven could read, at least single

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(1) Zeitschr. f. Nervenheilk., 1899, XV, p. 319.

words, written or printed; but not one of the seven could execute a complicated written command comprising five elements. Evidently it is not a question of word blindness, but one of intellectual acumen. As Marie points out, intellectual deficit manifests itself in three ways: First, in the deficit seen in cases of arrest and imbecility; secondly, in cases of mental disease in which dementia is present; and thirdly, as seen in aphasia. Clearly the deficit in aphasia is different from that observed in either of the other states, but it is none the less an intellectual deficit. The deficit in aphasia differs from the deficit in arrest and dementia in that it is lacunar, involving one function or a closely related group of functions, while in arrest and dementia, the deficit is more or less generalized.

Dercum next attempted to arrange the fourteen aphasics according to the presence or absence of anarthria. When one contrasts the utterance of an aphasic with that of a normal person, the great departure from normal articulation is at once apparent. In this attempt at classification Dercum failed. The motor speech difficulty appeared in each case as something added to the essential symptoms of the aphasia; an anarthria added to the special intellectual deficit.

"It would seem that Marie's contention that aphasia is a unity is well borne out by the clinical facts. The determination of the value of Marie's claim that all aphasia is due to an involvement of the zone of Wernicke, and that all of the motor speech phenomena are due to involvement of the zone of the lenticular nucleus, and that lesion of the third left frontal convolution has nothing to do with the motor phenomena, is a matter for future pathologic research. Marie's contention that motor phenomena may be produced by the involvement of the inferior longitudinal bundle and the isthmus which connects the zone of Wernicke in the region of the basal ganglia bears a strong probability of truth.

"It must be conceded that Marie's view is very attractive. It would indeed appear that aphasia is in reality a unity, that it consists not in a word deafness, not in a word blindness, not in an inability to utter words, but in a difficulty or impairment of the faculty of comprehending language, and that it is due to lesion of the zone of Wernicke.

Whether in a given case anarthria is also present depends entirely on the concomitant involvement of the inferior longitudinal fasiculus or isthmus in its passage to the lenticular nucleus and involvement of the lenticular nucleus itself. Whether in a given case, on the other hand, alexia is also present, depends on the involvement of the inferior longitudinal bundle posteriorly in its passage to the visual centers of the occipital lobe."

Charles L. Dana[1] discusses the *cerebral localization of aphasia* and its classification on an anatomic basis. He admits that the discussion of aphasia aroused by Marie is the exciting cause of the appearance of his paper, but that the subject-matter was prepared three years ago, so that in so far as he agrees with Marie, it is independent of any suggestions by that distinguished observer. To establish a diagnosis we first determine whether a defect of language function, or aphasia, is present. We have to exclude simulation, hysteria, mental disease and defect of the organs of speech. This leads to certain tests, as the following: 1, Voluntary speech; 2, exclamatory speech; 3, responsive speech; 4, associative speech, such as counting, saying alphabet, repeating familiar lines; 5, quality of speech, such as paraphasia and confusion; stereotypy, jargon; 6, repeating spoken words; 7, indicating the number of syllables in a word (to show that the patient knows that it is a word); 8, writing; 9, writing to dictation; 10, copying; 11, singing (humming tunes); 12, gesturing; 13, understanding gestures; 14, understanding spoken words, sentences and complex directions; 15, naming things seen, also felt, heard, touched, smelled, or tasted; 16, naming abstract things and qualities; 17, recalling to mind objects named; 18, reading understandingly, aloud or silently; 19, reading letters or numerals; 20, reading aloud without understanding; 21, praxia or knowledge of the use of things; 22, general intelligence.

In going over these tests we get various special symptoms by which we qualify the aphasia. The principal symptoms are these: (1) Aphemia, or inability to speak; (2) paraphasia, or jargon speech; (3) agraphia, or inability to write; (4) amusia, or inability to sing; (5) anomia, or inability to name objects; (6) word-deafness, or inability to understand words or sentences; (7) alexia, or inability to read words; (8) mind blindness, or inability to recognize things seen; and (9), visual apraxia, or inability to recognize the use of things. Of less importance are difficulties in copying, repeating, and understanding gestures, etc.

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(1) New York Med. Jour., Aug. 10, 1907.

In examining a large number of aphasics, Dana finds four groups: (1) Frontal lobe aphasia, or fronto-capsular aphasia (aphemia); (2) occipital, or parieto-occipital aphasia (pure alexia); (3) temporal, or parieto-temporal aphasia (the sensory aphasia of Wernicke); and (4) frontolenticular, or mixed aphasia. Of these groups (1), (2) and (3) are rare, and group (4) rather common. Most of this group are really due to lesions in the territory of the lenticulostriate artery, which supplies the corpus striatum, internal capsule, and the fibers converging into it from the third and second frontal gyri, and from the precentral convolution. In such lesions there is generally very complete aphemia, some alexia, and some difficulty in understanding language. With these symptoms is associated a decided hemiplegia without sensory symptoms. This is then clinically a mixed aphasia, and is anatomically a lenticular zone aphasia. It is called by Marie anarthria *plus* the aphasia of Wernicke.

"It is well agreed that there is a *zone of language,* involving the third frontal and adjacent part of the intermediate psychic area of Campbell, the first and part of the second temporal in its posterior three-fifths, some of the supramarginal and angular gyrus, and the second or perhaps more of the occipital. Beneath these areas are association tracts, including especially the superior and inferior association bundles. These association tracts must all pass through some part of the so-called lenticular region or zone. There is a good anatomic reason why this region then should be so extremely important in aphasia, as it includes the pyramidal tract, the association tracts, and an important supplementary motor center the lenticular nucleus.

"The cortical motor centers for the movements of the organs of speech are not part of the language zone. If they are destroyed we do not get aphasia. In some way, however, they are coördinately and effectively innervated in speech. There must be some organ or cortical area in which this is done. It seems to me that in accordance with the older views, it must be the intermediate psychic area of Campbell lying in the second and third frontal convolution.

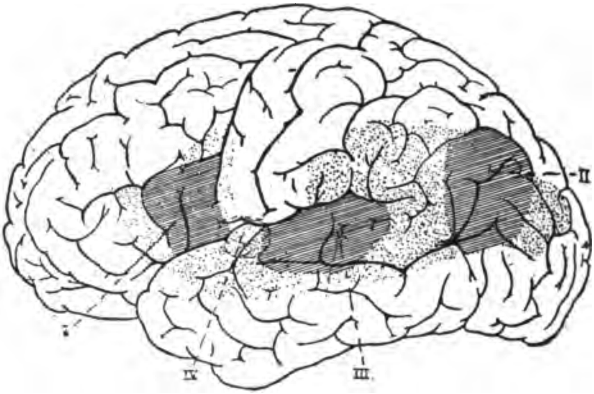

Fig. 3.—Diagram showing the zone of language and the localization of the four types of aphasia. I. Frontocapsular aphasia, characterized by aphemia, with ability to read, write and understand; hemiplegia present. II. Parietooccipital aphasia, characterized by alexia, with ability to write, speak and understand; hemianopsia present. III. Temporal or temporoparietal aphasia, characterized by anomia, some mind deafness, paraphasia, much agraphia and alexia; no hemiplegia, but some astereognosis, anesthesia and ataxia present. A small lesion may cause only optic aphasia. IV. Frontolenticular aphasia, the common mixed aphasia, characterized by aphemia with much agraphia, alexia, mind deafness and deafness. Hemiplegia present.

"Marie has achieved merit in abolishing the word 'motor' as applied to aphasia. Others before him have declared that all aphasia is sensory. I would go as far as this and apply the term 'sensory,' if it is so desired, to the frontal area also, where are stored up the associations and memories concerned with the function of articulation. It does not seem to me that Marie has proved his case with regard to the Broca convolution, *i. e.,* that it has nothing to do with the language zone. It is a cortical area concerned in the elaboration and coördination of the movements of speech. It is not a motor area, but a psychic one."

N. Vaschide(1) has investigated the intellectual power of aphasics and finds their memory and mind much diminished. This is in accordance with the contention of Marie.

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(1) Rev. neurolog., June 15, 1907, p. 543.

Variot and Lecomte[1] report a case of *congenital word blindness,* for which they propose the term *typhlolexia congenita.* The patient was a boy of 13 years, apparently normal in intellectuality, except that he could not read or understand printed words, despite the fact of excellent and numerous teachers. His visual memory for other things than printed words was good, and on an outline map he could draw in all the rivers and mountains and locate the leading cities without trouble. Evidently his reading-center is insufficiently developed functionally. Like other cases, he was more strongly *auditif* (as Charcot called it) than *visuel,* and was taught by having things repeated to him. His case, reported in full, needs no further attention.

As bearing on the question of aphasia, L. A. Amblard[2] reports a case showing mitral insufficiency and *left-sided hemiplegia with aphasia in a left-handed patient* also suffering from hysteria. He admits the condition of hysterical spasmodic narrowing of the mitral valve, but regards his patient as showing organic disease of the heart. The case was in Huchard's service. This curious association of aphasia with left-sided hemliplegia in a left-handed patient is contrary to all diagnostic rules. Similar cases have, however, been reported.

ARTERIOSCLEROSIS.

Joseph Collins[3] discusses the relation of arteriosclerosis both to disease and to disorder of function of the nervous system, his study of the subject being based upon 800 cases. Of these, 206 cases were definitely classified as arteriosclerosis of the nervous system, while in 595 others arteriosclerosis was found superimposed on some other definite disease. "Arteriosclerosis may cause: (1) Diseases of the nervous system that are well-defined clinical entities, and are therefore described as definite diseases; (2) it may superimpose definite or indefinite symptoms on any disease, incidentally altering the clinical feature of the disease; (3) it may give rise to symptoms which are so disparate that it is difficult to group them under any one heading, whence they are therefore often considered to be functional, neurasthenic, or hysteric; and (4) it may cause symptoms almost identical with those of well known diseases."

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(1) Gaz. des Hôpit., Oct. 30, 1906.
(2) Gaz. des Hôpit., March 19, 1907.
(3) Southern Med. and Surg., July, August, September, 1906.

Symptoms. The chief manifestations that cerebral arteriosclerosis produces are: (1) Insomnia and dysomnia; (2) headache and cephalic paresthesia; (3) vertigo; (4) the brain tumor complex; (5) the neurasthenic symptom complex; (6) epilepsy, focal and general; (7) amnesia and dementia; and (8) apoplectic symptoms. Each of these is discussed in detail by the author except the last two. Illustrative cases are introduced under each general symptom. Very interesting are the cases with the brain tumor complex of symptoms, three of which are recorded; and Collins affirms that in every case in which symptoms indicative of brain tumor occur in an individual above 45 years of age, cerebral arteriosclerosis should be suspected in the absence of implication of the optic nerves. Collins has seen seven such cases, and in three of them the diagnosis was confirmed by subsequent autopsies.

SENILITY.

M. A. Léri[1], at the meeting of the alienists and neurologists at Lille in August, 1906, presented the subject of the senile brain. Old age is an expression for the later periods of life and refers to a normal condition; senility is an expression for a pathologic condition not necessarily associated with age, sometimes coming on fairly early in life. Senility and old age are not, therefore, synonyms. Depending on the age of onset and its difference in the whole organism or over one or more organs, senility may be said to be precocious or late, general or local. There is no organ which is senile which does not show pathologic changes; and this, of course, applies to the senile brain.

The senile brain is essentially an atrophic brain, more or less sclerosed, and arteriosclerotic. Its weight and its volume are much diminished. The atrophy involves chiefly the anterior portion. The convolutions are slender, separated by deep and broad sulci. The pia mater is often thickened and meningitis is frequent; on the other hand, it is sometimes considerably thinned and fenestrated. The ventricles are dilated by reason of the cerebral atrophy, and their capacity may be doubled; their walls show a granular appearance. The corpus callosum is thinned, especially at its central part. Even the gray basal nuclei are atrophic.

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[1] Gaz. des Hôpit., Aug. 9, 1906.

Often sections of the brain show a vacuolated appearance, the lacunæ of disintegration of Marie, and sometimes one finds the so-called "worm-eaten" appearance of the cortex. The cerebellum may be atrophic, but is relatively less so than the cerebrum. Microscopically the nerve cells appear diminished in number and volume. The cellular atrophy may be a simple one with rarefaction of the bodies of Nissl, or with a heaping up of the pigment. The dendrites diminish in volume and number, the protoplasm stains badly. Surplus of yellow pigment granules is almost an absolute rule in senile cortical cells. The fibers of the corona radiata, the transversal, and especially the tangential fibers are all rarified, particularly in senile dementia.

The disappearance of the medullated fibers explains why the atrophy of the white matter exceeds that of the gray; the peripheral part of the white matter becomes gray by the absorption of the myelin sheath, so that the cortex augments in thickness at the expense of the subjacent white matter. The increase of neuroglia cells is only apparent, not real, the apparent increase being due to an actual decrease of the nerve-cells proper; yet an increase of neurogliar fibers causes the sclerosis found. Although such sclerosis is always diffuse, it affects certain places especially, such as the sub-ependymal, the perivascular and the subpial areas. The arteriosclerotic changes do not always harmonize with those of the base; they usually consist of an arteritis which is total, but they may consist of an endarteritis, a periarteritis or a mesarteritis. Colloid degeneration, fibrohyalin degeneration, and calcareous degeneration are rare. The lesions of the capillaries are little known. Fatty degeneration seems neither very frequent, nor proportionate to the sclerosis of the small arteries. Focal lesions in cerebral senility are determined chiefly by vascular changes, of which the following are the most important:

1. Perivascular and paravascular sclerosis, that is to

say at a distance, in territories doubtless badly irrigated.

2. Miliary hemorrhages, independent of miliary aneurysms.
3. Small multiple foci, of incomplete softening, such as occur in "arteriosclerotic atrophy of the brain, chronic subcortical encephalitis," or "senile cortical destruction."
4. Foci of lacunar disintegration.[1]
5. Foci of the so-called "worm-eaten" state.[2]
6. Large foci of softening and of cerebral hemorrhage, in no way differing from those of normal old age.

Symptomatology. The affection by which senility is most commonly evidenced is cerebral arteriosclerosis. Its principal symptoms have a paroxysmal character, being veritable attacks of cerebral intermittent claudication, and consist of vertigo, headache, noises in the ears, somnolence, disturbances of character, rapid mental and physical fatigue, amnesia, disturbances of speech, transitory aphasia, hemiparesis, short apoplectiform attacks not followed by hemiplegia, etc.

Cerebral arteriosclerosis may assume, at the beginning, the picture of neurasthenia. Hemiplegias, sudden and complete, like those due to ordinary cerebral hemorrhage, are no more frequent than they are in normal old age. They end fatally in the great majority of cases before the stage of contractures is reached. The so-called hemiplegia of the aged is due most often to lacunæ of disintegration. The paralysis comes on generally as the result of a very slight stroke, is very incomplete and transitory, as a rule affecting the legs. The gait is therefore disturbed, the steps being very short, and the feet dragged along. Dysarthria, dysphagia, spasmodic laughing and crying, often give to the patient an appearance of pseudobulbar paralysis. Senile epilepsy may occur and show all the clinical varieties of juvenile epilepsy, with or without symptomatic modifications. If it occurs it indicates either syphilis or arteriosclerosis.

From the psychiatric viewpoint, simple or deliriant dementia holds first place in cerebral senility; but a whole series of transition forms between perfect conservation of the intelligence and senile dementia occurs. Most senile

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(1) See Practical Medicine Series, 1903, Vol. x, p. 156.
(2) See Practical Medicine Series, 1905, Vol. x, p. 174.

sufferers present more or less marked enfeeblement of the intellectual faculties, first evidenced by a lack of memory of recent events and a diminution of creative imagination. Next, there are modifications of character, notably a loss of the ordinary affection for other members of the family, selfishness, avarice, excessive emotionalism over the little facts personally concerning them, a tendency toward hypochondriasis and delusions of persecution, and lastly a weakening of the syllogistic faculties. Senile dementia is only the exaggeration of the loss of all the faculties, a return to instinctive action, with conservation of automatic acts more or less complicated. The delirious ideas, accompanied or not by sensory troubles, are always inconsistent and unsystematic. Irresponsibility is evident in all their acts.

CEREBRAL BIRTH PALSIES.

Surgical Treatment. Charles E. Atwood[1] writes a critique of the recent surgical treatment of cerebral birth palsies. Prior to 1905 operations on the brains of infants suffering from this malady had never been recorded. Cushing's work is doubtless familiar to many, but its importance increases with a realization of the relative frequency of hemorrhage as a cause of infantile cerebral palsy. Starr, in his personal experience, found 45 per cent. of all cases of infantile cerebral palsy dated from birth, and an analysis of 341 cases studied at the Vanderbilt Clinic by Atwood shows 27 per cent. so dating.

No one seems to have given proper attention to the character of the brain injury which occurs simultaneously with the surface hemorrhage; but autopsies have shown that subsequent to hemorrhage we may have either a meningoencephalitis combined with lobar sclerosis; or a pseudocyst, a pseudoporencephaly, or cerebral atrophy; areas of softening, cicatrices, thinning of the cortex, or intense hydrocephalus. Both the psychic and the motor zones of the brain, as well as sensory areas, may be involved.

Dr. Cushing's experience teaches us, in the first place, that the condition of meningeal hemorrhage in the new born is capable of diagnosis and localization, although localizing symptoms do not appear sometimes until several days

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(1) New York Med. Jour., Jan. 19, 1907.

have elapsed. Second, that the operation is well borne. Third, that a hopeful result is obtainable sometimes in an apparently hopeless condition. The diagnostic symptoms produced may be divided into those which are general and more or less transient; and those which are localizing and tend to be permanent. The bulging fontanel, which in many cases does not pulsate, the asphyxia and the slowing of the pulse we would speak of as general symptoms; the convulsions are often localizing, as they are frequently unilateral. There is often stupidity in nursing or difficulty in swallowing. Localizing symptoms in the shape of muscular twitchings, rigidity or paralysis may be present; or conjugate deviation of head and eyes to the side opposite the lesion. An affected arm or leg may not be moved. Both legs may be rigid and exhibit convulsive movements, showing a double sided lesion of the leg centers.

Hemorrhage occurs most frequently over the motor areas. If unilateral we have but the opposite side affected; if bilateral both legs are affected mostly and one or both arms to some extent, and the trunk centers, lying near, may be involved. If an operation for removal of the hemorrhage is not performed the patient dies; or, the blood is slowly absorbed. In the latter event the initial brain damage is augmented and becomes permanent before really a general improvement may take place.

In conclusion, it seems reasonable to say that immediate surgical intervention in intracranial hemorrhages of the new born is justifiable in properly selected cases, as offering hope where no immediate hope seems otherwise to exist, and also from the standpoint of mitigating distressing sequelæ. Early operation is found to be well borne. The blood is found coagulated as in similar conditions in the adult. Quoting from a personal letter from Dr. Cushing: "Success depends on two things: First, the early and prompt recognition of the condition by the obstetrician; and second, the delicacy of manipulation and painstaking methods of the operator.

APOPLEXY AND HEMIPLEGIA.

Sudden Ceabral Lesions. The diagnosis and immediate treatment of sudden cerebral lesions is the title of a clinical lecture by Sir William Gowers[1] The usual sudden lesion causes hemiplegia; rarely there is diplegia, or possibly merely a loss of speech, impairment of articulation, or hemianopia. An organic lesion of the brain of sudden onset, coming on in a few minutes or a few hours, is a vascular lesion, and in the vast majority of cases an arterial one, due either to its rupture or its blocking. The several arterial states causing these lesions differ in frequency in the first and second periods of life. In later life they are commonly due to degenerative changes in the walls of the vessels; in the former, to other disease. The causes that are active in the first half may, however, extend on into the second. Most sudden lesions occur in later life.

The first question, therefore, in sudden hemiplegia is the age of the patient. The degenerative period of life may be said to begin at 50, but it may begin earlier in those showing an inherited tendency to early arterial degeneration. The first point to be noted, then, is the state of the pulse and heart. High tension of the pulse and a strongly acting hypertrophied heart point to hemorrhage, and a soft feeble pulse and a dilated heart are the indications of thrombosis. Distinct degeneration of the arteries of the arm is a little in favor of atheroma, but they may be soft when the cerebral vessels are affected. Tortuosity of the temporals is no index to the state of the arteries within the skull. If a heart, whether weak or strong, presents also a valvular murmur, the possibility of embolism arises.

Preceding cerebral attacks if slight, and especially if repeated, are strongly in favor of thrombosis and softening. Warning symptoms during the few days before the attack are an important indication of thrombosis. They consist of tingling in the parts afterwards paralyzed, transient weakness, brief loss or impairment of speech, vertigo, and the like, and are probably due to impairment of circulation in the narrowed vessel. Moreover, in thrombosis, a condition of fatigue, exhaustion, or depression is often to be traced just before the attack. Physical fatigue is a frequent antecedent,—a tiring journey, a long walk, a day's

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(1) Brit. Med. Jour., July 6, 1907.

shopping. In other cases the attack follows mental distress, a sudden shock or a distressing grief.

The onset of symptoms is much more frequent in sleep than is the case in hemorrhage. Loss of consciousness occurs in either thrombosis or hemorrhage, but more readily in the latter. Consciousness in thrombosis is lost only when a very large vessel like the middle cerebral artery is occluded, while blockade of one of its branches usually does not cause such loss. Since the clot more often forms in the branch than in the main artery, a loss of consciousness is comparatively infrequent.

Convulsions at the onset are more frequent in softening than in hemorrhage, at any rate if they are unilateral; and their significance is greater if they are partial and repeated, and attend imperfect hemiplegia, because such symptoms are generally caused by partial disease of the motor cortex; and a small cortical lesion is much more frequently due to thrombosis in a terminal branch of the middle cerebral artery than to hemorrhage. Locality, too, is significant. Hemianopia, alone or with hemianesthesia, suggests softening, because thrombosis in the posterior cerebral artery is more common than hemorrhage. The same is true of lesions of the pons. In hemorrhage into the pons consciousness is early lost, and may be attended with a rapid and fatal hyperpyrexia. Coma also attends thrombosis of the basilar artery, but it usually comes on gradually, as a deepening stupor, always of the gravest augury in such an association.

"Early signs of considerable irritation are in favor of hemorrhage. The laceration of the nerve elements has this effect in a marked degree. It causes loss of consciousness by upward irritative inhibition, and sometimes, though not often, it causes convulsions. It also, acting downwards, may cause early and great rigidity, but sometimes initial flaccidity of the paralyzed limbs, even with loss of the knee-jerk during the first twelve or twenty-four hours, while on the second day it becomes excessive, with foot clonus. Early rigidity with clonus may be met with during the first few days in softening, but the irritative inhibitory relaxation is probably confined to cases of hemorrhage. Considerable depression of temperature during the first day—two, three, or four degrees below the normal—is a sign of hemorrhage.

A rise of temperature to about 100° F. about the third day is more common in softening.

"'Ingravescence,' the steady increase of coma from a slight to a profound degree, when it takes place in a few hours, signifies hemorrhage. When it occupies one or several weeks it is usually the result of spreading thrombosis from extensive atheroma. A peculiar state of deepening stupor, from which at first the patient can be roused, often attends thrombosis of the basilar, other evidence of which can usually be found. A rapid rise in temperature to a 'hyperpyrexial' degree sometimes attends ventricular hemorrhage, but is especially suggestive of a lesion in the upper part of the pons, which may be of any character. It gives great significance to anomalous ocular symptoms; usually it means death, but I have seen a patient who survived a temperature of 110.8° F., apparently due to embolism of the extremity of the basilar.

"To sum up. The patient is in the degenerative period of life, or has advanced kidney disease. The indications of hemorrhage are few: hypertrophy of the heart and a tense pulse; an onset attended with loss of consciousness, either profound or of long duration, or disproportionate to the degree of paralysis, and there is often a considerable initial depression of temperature, and sometimes complete flaccidity of the paralyzed limbs. To these symptoms the absence of the indications of thrombosis may be added.

"These indications are more numerous: A feeble, soft pulse, and weak, often dilated heart, sometimes irregular; previous attacks, especially of slight degree; transient premonitory symptoms, often headache; the onset often deliberate, with loss of consciousness if the paralysis is severe. Convulsion at the onset is not of much significance, but, later, is in favor of softening; and so with aphasia, which, if lasting, indicates softening. So does much impairment of articulation or swallowing, suggestive of a pontine lesion; and also hemianopia, showing that the mischief is in the region of the posterior cerebral artery.

"In the immediate *treatment,* some measures may be adopted in each lesion. A gouty state is common, and generally presumable, because age-imperfection of the organs generally leads to an approximate condition, and justifies some lithia or potash, combined with a diuretic, nitrous ether and spirits of juniper, to which some digitalis may be added—more if thrombosis is suspected than if hemorrhage is probable. The opposite rule should obtain with an aperient. If a clot has formed, only gentle action of the bowels should be secured; if a vessel has ruptured, an active purge is wise, such as croton oil, to lower the blood pressure. Blood should be drawn to the extremities.

"If there is reason to regard the lesion as thrombosis, little more can be wisely done. We have no practical agent to reduce the coagulability of the blood. Citric acid is said to have this effect, and may be given as lemon juice in plenty of water. When hemorrhage may be confidently diagnosed, the treatment should be such as to increase the tendency of the blood to clot. Salts of calcium have this effect, and may be given in whatever form can be most promptly obtained. Arsenic, by hypodermic injection, has a similar effect, and 1-5 grain of the arsenate of sodium may be given in a few drops of water. There is no reason to believe that small doses of iodid of potassium, which are constantly given at the early or later stage, exert the slightest influence.

"But one measure is imperative in every case of either kind—physicial tranquility. It should surely be superfluous even to mention this, but experience shows, too often, that it still needs to be insisted on. Even in the slightest case, if the patient must be moved, it should be as little as possible, and he should be absolutely passive."

Predegenerative Lesions. During the first part of adult life, up to 45 or 50 years, most sudden cerebral lesions are due to arterial closure, either as a result of embolism or of syphilis. Of course embolism or syphilitic disease of the arteries may occur subsequent to the age of 50 years, in which case diagnostic difficulties are encountered. Each may occur, too, in earlier life, but arterial disease from inherited syphilis is very rare. As a rule the diagnosis of embolism presents a simple problem.

With rare exceptions the source of the embolus is the left side of the heart. It is useful to remember that the mitral orifice is far more frequently the source of the plug than the aortic, and that mitral stenosis is specially prone to give rise to detachable vegetations. Perhaps this is because the blood flows more slowly through the mitral orifice during diastole, when vegetations easily increase in size, than it flows by the aortic cusps after the powerful systole of the ventricle. A very important fact is that in embolism there are no related preceding symptoms. The state of the vessel is normal until the embolus is brought by the blood current. Suspicion should therefore be felt as to the nature of any case, apparently embolic, in which premonitory symptoms were distinct, especially unfamiliar headache. Embolism may also occur in the degenerative period of life, at any age, and the diagnosis is then more difficult or even impossible.

Embolism less commonly results from degenerative changes in the valves than from actual endocarditis, but it does occur from them. No features of the attack are of special value in the diagnosis. There is a slight excess of lesions on the left side of the brain in embolism, and also considerable aphasia is more frequent, while pyrexia during the first week is more pronounced, but the preponderance of these signs, is not enough to have diagnostic value.

Of the *treatment* of embolism there is little to say. "Whatever tends to excite the heart must be carefully avoided and its action steadied and strengthened, with the object of hindering the extension of the clot and of promoting the establishment of any collateral circulation that may be possible. It is believed that sometimes a soft plug breaks up into particles small enough to pass away, but this can only be possible during the first few hours, and no means of promoting it are known, nor would any attempt be wise. Occlusion, once established by distal clot, must endure, and so must its effects, unless the blood supply can be renewed by collateral communication."

Syphilitic Arterial Disease. This condition in the predegenerative period of life leads to thrombosis. Although the primary condition is a slow, gradual process, the actual closure is sudden or rapid, because the flow of blood through the narrowed passage becomes less and less, until at last clot suddenly occludes it. The thrombus probably begins to form just beyond the narrowing, where the flow is reduced to the middle of the lumen, and at the sides the blood must be almost stationary. It must be so, whether the cavity of the main artery is thus reduced, or the orifice of a branch at the spot diseased is progressively narrowed. It is important to realize this fact, that the vessel is closed by a process independent of the syphilitic disease which causes it, because the clot cannot be influenced by the treatment that may lessen the disease in the arterial wall, and an artery once closed is closed forever.

This cause may be assumed, in any case during the predegenerative period without heart disease, with confidence when previous syphilis is known, and with probability whenever it is possible. It is rendered possible by any risk of infection. In most cases the absence of any other causal condition, and the fact of certain or presumable syphilis, suffice for the diagnosis and render the special features of the attack of subordinate importance. If a cardiac murmur is found, remember that aortic disease is not a common cause of embolism, and is sometimes caused by syphilis. The most important evidence is afforded by premonitory symptoms, especially headache, during a week or more before the onset. Sometimes there is a history of mental dullness, giddiness, or of numbness, tingling, or passive weakness in the limbs afterwards paralyzed. Some symptom of this character is to be heard of in more than half the cases; showing as it does a morbid state before the attack, it is an almost conclusive distinction from embolism. No features of the onset present a special character or frequency sufficient to be of strong weight in the diagnosis. It occurs during sleep in a third of the cases. Loss of consciousness is rare. Transient loss can, of course, only be recognized when the onset is in the waking state, but of these it was described only in 12 per cent. Neither convulsion nor considerable aphasia is common, but either may occur. Compared with other morbid processes, syphilitic disease has a disproportionate tendency to affect the basilar artery sufficient to be of some diagnostic significance in a doubtful case.

"When the past history of a patient is known, the length of time that has elapsed since the primary disease is an element that may properly be allowed some weight. After what interval is arterial disease intrinsically improbable? I have found that one-half of the cases occur during the first five years after infection,. and a quarter during the second five years; during the first fifteen years, seven-eighths of the whole occur, and after twenty years examples are doubtful. Hence the probability is low after fifteen years and negligible after twenty. But we may not draw any similar inference from the age of the patient. Syphilis may be acquired at any age, and in later life the tendency to secrecy is far greater than in earlier years, and is often, indirectly, the cause of death."

Treatment. Iodid should be promptly given in such a case. Twelve or fifteen grains may be given every three hours for the first two days, then every four hours, and reduced to three times a day at the end of a week. Larger doses are unwise, because they may increase the coagulability of the blood. One may afterwards employ a course of mercury, but lesions of the type of that in the arterial walls seem to yield very promptly to iodid, and the system can be saturated with iodid as it cannot with mercury. Of course, nothing can influence accomplished occlusion. A thrombosed artery can never again become permeable. Improvement generally follows, just as it does in embolism, because the initial symptoms are more severe and extensive than those that are permanent. The reason for energetic treatment is that the disease generally exists in other arteries also; its amount cannot be known, and some other vessel may be on the verge of closure. Equally important is the prompt treatment of premonitory symptoms.

Hemiplegia in Uremia. Cases of hemiplegia occurring in uremia are discussed by John H. W. Rhein[1], who has examined pathologically seven cases in which no gross anatomic changes were found. The mechanical theory, which explains the paralysis of uremia as due to cerebral edema; the toxic theory, which holds that the cerebrospinal fluid is toxic and elective for nerve-cells; the theory of cortical malnutrition, which regards the paralysis as the effect of obliteration of vessels supplying the cortex; and the theory that uremic hemiplegia is due to atheroma of the cerebral arteries are all regarded as insufficient and unsatisfactory.

In the seven cases examined by Rhein the findings of six warranted a diagnosis of cerebral syphilis, while the remaining case showed a lesion, although not entirely char-

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(1) Jour. Am. Med. Assoc., Nov. 24, 1906.

acteristic of syphilis, yet pointing toward that malady. In this last case there was much arterial change, and the perivascular spaces of the paracentral cortex were markedly distended. In all seven cases there were found microscopic areas of softening, without evidence of gross cerebral disease. Even microscopically the softened areas may be readily overlooked. Though these are very small, probably only the size of a point of a pin, it is conceivable that if numerous, they could bring about some distinct alterations in the functions of the cortical nervous tissue, sufficient to cause paralysis.

To Rhein it seems highly probable that these lesions are responsible for at least some of the cases of so-called uremic hemiplegia. Rhein[1] previously described these necrotic foci in connection with double hemiplegia. They were present in that case in the paracentral region, and were associated with disease of the blood-vessel walls, round-cell infiltration of the pia, and other syphilitic lesions. While it is well known that areas of softening occur in various portions of the brain, they are usually extensive enough to be recognized by the naked eye. These findings must modify the views previously held about uremic hemiplegia and other forms of hemiplegia in which there is no gross cause demonstrable at the autopsy.

Rhein is forced to the conclusion that uremic hemiplegia is not due to any one cause in all cases, but may be due in a given case to one of a number of causes; in some cases to autointoxication, in others to edema or arteriosclerosis; in still others to the presence of microscopic areas of necrosis just described. Another cause for these paralyses is syphilis. Doubtless the perivascular distension plays some part in the causation of the paralysis, probably by causing pressure on the cells of the cortex,—a pressure which varies directly with the amount of leakage from or absorption of the fluid into the perivascular spaces.

Involvement of Muscles of Mastication. In general it is taught that in hemiplegics the different movements of the jaws are always maintained. C. Mirallié and A. Gendron[2] dispute this statement. The muscles concerned, the temporal, masseter, and two pterygoids, may be easily tested.

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(1) Am. Jour. Med. Sciences, May, 1906.
(2) Rev. neurol., Dec. 30, 1906.

It will frequently be found that the power of these muscles is diminished on the hemiplegic side. Many hemiplegics cannot retract the lower jaw at all, and sometimes forward projection is impossible. Fourteen illustrative cases are given.

ENCEPHALITIS.

L. Murat[1] reports a case of primary acute encephalitis. This affection, of which several cases have been reported in Germany, was first reported in France by Raymond and Cestan in 1904, except that Murat had chosen this disease as the title of his Lyons thesis in 1897. Since that date Murat has collected about 100 cases, almost all of them reported outside of France. In the present communication a typical case of acute encephalitis is described, secondary doubtless to la grippe. As Raymond[2] has recently written, up to the present time the dogma of vascular obstruction has too exclusively prevailed as the cause of cerebral softening. There are cases proving incontestably that there is such a thing as inflammatory softening; true acute degenerative encephalitis.

SINUS THROMBOSIS.

A. Wimmer[3] reports an interesting case of a man, 52 years of age, brought to the hospital with the diagnosis of apoplexia cerebri made in the clinic. On the day he was received, while he was at work, he was suddenly paralyzed in his left arm, and was seized with a shaking extending over his whole body. He did not lose consciousness, did not fall, but on the contrary walked home with the assistance of companions. Towards evening jerkings appeared in the right half of his face, and he became a little stuporous. After arriving at the hospital he had three attacks of clonic spasms in his left arm, lasting each about half a minute. There were found a paresis of the right facial nerve, complete paresis of the right arm, and partial of the right leg. The reflexes were all exaggerated, especially on the right side. There was no deviation of head or eyes. In four days he died.

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(1) Gaz. d. Hôpit., Dec. 4, 1906.
(2) Arch. de méd. des enfants, November, 1906.
(3) Berlin. med. Woch., Nov. 12, 1906.

Evidently the usual capsular hemorrhage was not to be thought of. Because of the more or less episodal character of the onset, and especially because of the Jacksonian type of the symptoms of irritation, the seat of the lesion was ascribed to the cortex. The age of the patient and his rigid arteries led to the diagnosis of possibly an embolus with consequent thrombosis in the territory of the left middle cerebral artery. Tumor of the brain and meningitis were not to be thought of, and there were no prodromal symptoms suggestive of an acute hemorrhagic encephalitis. Postmortem examination disclosed a complete thrombosis of the superior and inferior longitudinal, the cavernous, and transverse sinuses. This had extended out into the veins of the pia, especially on the left side. The pia itself was somewhat edematous. The arteries were intact. The etiology was unknown, but it may be said that there were tuberculous cavities in both lungs with a fibrinous pleurisy, and there was fatty degeneration of the liver, and a few tuberculous ulcers of the cæcum. Possibly the case belongs to the "marantic" group of sinus thromboses.

C. Eugene Riggs[1] reports a case in which symptoms simulating brain tumor were found to be due to the obliteration of the longitudinal, lateral, and occipital sinuses. The symptoms were choked disk, vomiting and headache, together with other symptoms of slight localizing value. Twitching occurred late in the case on the right side of the neck, in the sternocleidomastoid and platysma myoides muscles. There was sometimes an accompanying tremor of the chin. Postmortem examination showed the sinus changes above mentioned. There was no apparent etiology for the obliteration found, other than a severe fall, in which the patient struck on her head upon frozen ground, two years previously.

CEREBRAL ABSCESS.

Symptomatology. Charles Bourret[2] reports a case of cerebral abscess of interest because showing *hippus*. The patient developed an abscess of the temporal lobe secondary to chronic otorrhea with ultimate involvement of the mastoid cells. A week after the mastoid operation she began

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(1) Jour. Nerv. and Ment. Dis., April, 1907.
(2) Lyon Méd., June 24, 1906.

to vomit, and there was obstinate constipation, rise of temperature and slowing of the pulse to 48 or 50. At the same time hippus appeared; the left pupil became dilated and showed spontaneous and rhythmic movements of contraction and dilatation. These movements were not synchronous with either respiration or pulse. There was no hemianopia.

On opening and draining the abscess the hippus disappeared. When, a week later, secondary disturbances with signs of meningitis developed, the hippus again appeared. Interesting are the facts that the hippus involved the pupil on the side opposite the lesion; and that the hippus varied directly with the meningeal and cerebral disturbances. Hippus has also been reported in hysteria, hystero-epilepsy, epilepsy, chorea, general paresis, multiple sclerosis, and tuberculous meningitis.

L. W. Rose and R. G. Cook[1] report a case of cerebral abscess which is instructive in its *symptomatology*. The patient complained of distress in the abdomen on Aug. 8, 1906, vomited, and fell twice when walking. Three days later he had pain on the right side of the neck and shoulder. Three days after this he began to "seem different" to his friends, and he had pain in the left side of his head and face. On the following day he was confused, slow of speech, aphasic, stupid, and acted as though he were under the influence of some narcotic. The temperature was normal, and his pulse was 60. Subsequently the temperature rose to 99.2° F. and the pulse to 80. He showed motor aphasia, and agraphia, with partial apraxia; that is, he could not recall names of objects, except very imperfectly, he could not copy writing or printing, and he tried to light a cigar with the unlighted match, when he was given two matches, one of which he properly lighted. Yet he realized his aphasia and even joked about not being able to remember the name of the company for which he worked.

On August 16 it was reported that he had not been able to speak for several hours nor to move his right arm, and that he had passed no urine since morning, and that he seemed to have had pain in the head. The blood-count showed 14,760 leucocytes per cubic millimeter. On August

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(1) Buffalo Med. Jour., February, 1907.

17 he had a general convulsion. On the eighteenth he had several convulsions, one of them being Jacksonian in type, beginning in the right side of the face and extending to the right arm only. The diagnosis of abscess was made on the seventeenth with a correct localization in the arm-center, and at the posterior end of the left second frontal gyrus, and the contiguous portion of the ascending frontal convolution. Operation was performed, the abscess found, opened, and drained, but death quickly followed.

M. Loedlein[1] reports the case of a brain abscess *caused by streptothrix* in a man, 58 years of age. It was found in the anterior pole of the right temporal lobe, its size being equal to that of a dove's egg. Two other small secondary foci were also found, closely adjacent. In the abscess was found a streptothrix. Nothing could be found in the right ear in any way abnormal. In the lungs, however, extensive bronchiectases were found in both lower lobes, but they did not contain streptothrix. Careful examination of the brain indicated the embolic origin of the abscess, however. The streptothrix found was thought to be identical with that described by Eppinger. MacCallum has also found the same organism in post-operative peritonitis.

Diagnosis. Barton H. Potts[2] emphasizes the difficulty of diagnosis of brain abscess. Being an aurist, he is chiefly interested in those cases secondary to disease of the middle ear. "The cardinal symptoms of cerebral abscess are neuroretinitis or choked disk, subnormal temperature, headache, chill, vomiting, slow pulse, slow cerebration, stupor, high leucocytosis with high polymorphonuclear percentage, and cerebrospinal fluid free from pus. Cerebellar abscess frequently gives no characteristic symptoms.

"The diagnosis is best given in Dana's cerebellar seizure, viz., sudden unexpected attacks of extreme vertigo, roaring in the head, relaxation of the limbs, and falling to the ground in a semi-conscious state. * * * Unfortunately our cases do not present groups of symptoms sharply defined. The changes in the fundus of the eye are generally late; their absence, therefore, is not a help. The temperature is frequently subnormal, but sometimes does not fall

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(1) Münch. med. Woch., July 30, 1907.
(2) Amer. Jour. Med. Sciences, July, 1907.

below 101° F. The slow pulse is a reliable guide when present, but in some cases it never goes below 80, keeping in perfect accord with the temperature. The headache and slow cerebration are nearly always present, but stupor is a late symptom. Kernig's sign may be present, but is more generally absent; it is always constant in meningitis.

"It may be said that the localizing symptoms of brain abscess are practically never present if the abscess be small, and they are frequently absent, even when the abscess is of considerable size. Abscess of the frontal lobe is usually attended with no localizing symptoms, unless it is situated about the base of the third frontal or ascending frontal convolution. [Abscesses being subcortical, give rise in the motor cortex anterior to the fissure of Rolando, to the subcortical group of motor signs.—ED.] If the abscess is large the pupil of the same side is usually dilated; if small, the pupil may be sluggish and contracted.

"If a temporosphenoidal abscess be small there are usually no localizing symptoms; when large the adjacent centers and nerves are likely to be affected, either by pressure or inflammatory action. Hemiplegia on the side opposite the lesion is occasionally found in large abscess. Facial palsy may be caused by lesions in the tympanic antrum, or it may be due to a cortical lesion. In the former case the paralysis is complete; in the latter, the patient is usually able to move the eyelid. If due to antral lesion it is on the same side; if due to cortex involvement it is on the side opposite the lesion.

"Aphasia has been noted in abscess of the left temporosphenoidal lobe; its presence is a help in diagnosis, its absence has no significance. If it be sensory aphasia, word deafness, the lesion is probably in the posterior part of the lobe; motor aphasia, in the anterior portion. Paralysis of the third nerve is sometimes seen in large abscess of the temporosphenoidal lobe, and when complete the picture is characteristic; ptosis, external strabismus, and fixed and dilated pupil. The eyeball can be moved only outward and slightly downward. It is seen, therefore, that no symptom is pathognomonic; that diagnosis is nearly always difficult, and must be made by the general clinical picture. When meningitis and brain abscess are found together the diagnosis may be difficult or impossible."

HYDROCEPHALUS INTERNUS.

W. G. Spiller and A. R. Allen[1] report two cases of internal hydrocephalus, one resulting from occlusion of the aqueduct of Sylvius. Though often mentioned in textbooks, no references to cases are ever noted when this is mentioned as an etiologic factor. The case reported by Spiller was that of a woman of 62 years. At death it was found that the aqueduct was almost occluded, this occlusion being probably a congenital malformation because the cavity was well lined throughout by a layer of ependymal cells. To the naked eye the aqueduct seemed to be entirely occluded. The second case is reported as one of intense congenital hydrocephalus without occlusion of the aqueduct.

CEREBRAL TUMOR.

During the past year unusual attention has been bestowed upon the subject of brain tumor and the literature is exceedingly extensive.

Diagnosis. Walton[2] reports a case of frontal tumor diagnosticated as cerebellar. The early complaints were pain over the right eye and down the right side of the face and neck, loss of vision, and vomiting, for a year. The past two months there had been severe and constant pain in the occipital region and back of the neck, with drawing of the head backward, and to the left shoulder, the pain extending to the arm with subjective numbness. There had been hysterical attacks with screaming. There was unsteadiness on standing with possibly a tendency to fall toward the right. There was double optic neuritis, with marked projection of the left disk. The right pupil was larger than the left, both reacting to light. The patient answered questions in a fretful manner, indicating a change of disposition. There was no asynergia, but decided diadokokinesia in the left arm in which she was unable to make rolling movements. The tumor was found postmortem projecting into the cavity of both lateral ventricles on each side of the septum lucidum, just anterior to the caudate nucleus.

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(1) Jour. Am. Med. Assoc., April 13, 1907.
(2) Jour. Nerv. and Ment. Dis., June, 1907, p. 400.

H. H. Hoppe[1] reports a case in a young woman of 23 years of age showing a symptom-complex suggesting brain tumor, but ending in recovery. He also reports two other cases, now alive. The young woman died of pulmonary tuberculosis, and the postmortem examination showed no brain tumor present. These cases are like those reported by Nonne. Hoppe believes the symptoms in these cases to be dependent on one of the following conditions: 1. Serous meningitis producing acquired internal hydrocephalus. 2. Hemorrhagic non-purulent encephalitis with serous effusion. 3. Chlorosis. 4. Chronic cerebritis. Practically, we should not be too pessimistic in our prognosis of brain tumors, especially in cerebellar cases, until a considerable period of time has elapsed. Moreover, we should not too hastily advise operation.

William M. Leszynsky[2] discusses the diagnosis of intracranial tumor, reporting five illustrative cases. It is not always safe to assume the existence of a tumor from such general symptoms as headache, vomiting, or even optic neuritis, because they may be present, for example, in cases of chronic basal meningitis. "In doubtful cases the diagnosis must be made by exclusion. The presence of optic neuritis is indicative of an intracranial lesion, and at once eliminates the functional type of headache. Epileptic convulsions, occurring for the first time late in life (in the absence of chronic nephritis) should always lead to examination for evidence of cerebral tumor. More attention and consideration should be given to convulsive seizures limited to one side of the face or one extremity without loss of consciousness. Such attacks are too often ascribed to hysteria, but in the majority of instances are either of uremic origin or the result of cerebral disease, possibly tumor.

"The experienced neurologist often finds the symptoms of intracranial tumor so perplexing that it may be extremely difficult to decide whether they are due in greater part to complicating internal hydrocephalus, or to the direct effect of the tumor itself. Increase of cerebrospinal fluid is an almost invariable accompaniment of an intracranial growth. A positive differentiation between ac-

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(1) Jour. Nerv. and Ment. Dis., February, 1907.
(2) Med. Record, July 13, 1907.

quired internal hydrocephalus and cerebral tumor is often impossible.

"Again, cases occasionally occur which simulate the clinical picture of brain tumor involving either the cerebral hemisphere or the cerebellum, which terminate in permanent recovery, leaving atrophy of the optic nerves as the only residual symptom."

Walter Timme[1] reports a case of metastatic carcinomata in the brain and cord. The case is interesting because no diagnosis of the lesion in the brain was made before death. It was regarded as pure *hysteria.* The following were the findings: The entire spine was hypersensitive. At the levels of the fifth, sixth and seventh dorsal vertebræ the pain on pressure was excruciating, eliciting shrieks of pain. This one symptom remained constant. There was a band of anesthesia around the abdomen varying in width and in position daily. There was a loss of knee-jerks. The left eye showed some nystagmus. There was ptosis of the left lid, and a slight convergent squint. The tongue deviated markedly to the left, though the patient could somewhat correct this on trial. There was a slight thickness and slowness of speech reminding one of the speech of beginning bulbar palsy. Deglutition was slow and necessitated effort. On several occasions, the patient complained of a frontal headache, relieved at once by cold compresses. Almost all the usual symptoms of hysteria were engrafted on the foregoing; thus the globus, the insensitive corneæ, the craving for sympathy, the hysterical crying, all were present. Opposed to these positive signs and symptoms there were the following negative conditions: There was no ataxia. While in bed, the patient could move her legs and feet in any direction; the muscular, tactile and temperature senses also seemed perfect. Control of bladder and rectum was unimpaired. There was no tremor of hand, tongue or facial muscles. The mental condition was excellent, there being no aphasia, agraphia or other memory fault. There was no vertigo, no nausea and practically no headache.

Postmortem, a tumor was found on the inner surface of the dura pressing on the left cortex, above the Sylvian

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(1) Jour. Am. Med. Assoc., July 27, 1907.

fissure, on the inferior, middle, and anterior central gyri. A second larger tumor was found at the base of the brain, extending from the left frontal region to the orbital plate. In the spinal canal, from the fourth cervical vertebra to the cauda equina was an elongated metastasis, which compressed the cord. The nerve roots were involved, especially, in the upper dorsal region.

"The larger of the two tumors of the brain, namely, the one under the frontal lobe of the left side, must have interfered in some way with the higher functions of cerebration. I recall distinctly the odd wit and humor which, even during her greatest paroxysms of pain, the patient often indulged in, relapsing quickly to her former state. German observers have noted this condition in abnormal states of the prefrontal lobes and call it *'Witzelsucht.'* Did the marked hysterical exaggeration also arise from the same source?"

Unusual Tumors. *Enchondroma.* F. C. New and J. A. Macdonald[1] report a case of brain tumor, remarkable for both the rarity of its form of growth and the extreme size to which it grew. The tumor occurred in a patient who died in a hospital for the insane at the age of 43 years. His first illness was major epilepsy, attributed to sunstroke, coming on him in his sixteenth year. Ten years later his left eyeball became abnormally prominent. Four years later a right hemiplegia developed. Death was caused by an intercurrent pneumonia.

Postmortem examination disclosed a tumor in the left hemisphere weighing 575 grams (about 20 oz.), its size being 16 by 12 by 9 cm. (6½ by 5 by 3½ in.). It extended from the left frontal lobe backward, covering the left parietal and temporosphenoidal lobes, and replacing them. A second growth, 3 by 2 by 5.5 cm. (1¼ by ¾ by 2 in.) was found springing from the inner surface of the left squama, in front of the petrous portion of the temporal bone. Microscopically the tumor was found to consist of normal hyalin cartilage. Osseous deposits in the dura, or small osseous projections from the inner surface of the skull are not infrequent, but enchondromata are very rare. [The authors, state that no report has been discovered by them

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(1) Jour. Am. Med. Assoc., July 14, 1906.

of any intracranial growth of the size of the one they describe. Schlesinger in the *Neurologische Centralblatt,* 1895, p. 702, and 1898, p. 974, describes a case in which the entire right cerebral hemisphere was replaced by a gliosarcoma.—Ed.]

L. Pierce Clark and C. E. Atwood[1] report a case of *multiple enchondroma,* one of which grew from the sella Turcica and caused pressure on the pituitary body, the chiasma and crura. The patient was a boy, seventeen years of age. At four years a tumor was removed from the surface of the right sixth rib. Others subsequently appeared from time to time on the left wrist, on the upper end of each tibia, at the costal ends of all the ribs on the left side, etc. They ranged in size from a pea to an orange. The patient and his relatives declared that the tumors came and went spontaneously.

"Cases of compression of the brain or cord by enchondromata are extremely rare. We have not found a recorded case. The tumors are of slow growth and the duration of symptoms in our case, together with the fact that there is some improvement in the eye symptoms, lead us to hope that there may be some retrogression in the intracranial tumor, the same as has occurred in some of the tumors in other parts of the body."

Epithelioma. A primary epithelioma of the brain is reported by R. Cestan,[2] a very exceptional postmortem finding. The case was that of a lad of seventeen years whose history was given by Professor Raymond in his *Cliniques des maladies du systeme nerveux* (5th series, p. 139), and the tumor was located in the sella Turcica because of the edematous optic neuritis, amaurosis of the left eye, hemiopia of the right eye, anosmia of the right nostril, and left-sided hemiplegia with paresis of the right oculomotorius. However, there was no acromegaly. At the postmortem the tumor was found in the sella Turcica, and proved to be a primary affair, apparently cystic. Cestan had previously found such a primary epithelial tumor in the pituitary region in a man showing evidence of tumor at the base of the brain without acromegaly, much resembling this one now found. Micro-

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(1) New York Med. Jour., July 20, 1907.
(2) Gaz. d. Hôpit., Aug. 7, 1906.

scopic examination showed it to be an epithelioma. The pituitary body was found in structure to be wholly normal.

Localization of Tumors. *Frontal Tumors.* T. Grainger Stewart,[1] in an exceedingly well-written article, discusses the diagnosis and localization of tumors of the frontal regions of the brain. "The clinical picture of frontal tumor is characterized by the absence of signs pointing to involvement of other regions of the brain rather than by the presence of special focal symptoms. In many cases the cardinal symptoms of intracranial growth, headache, vomiting, and optic neuritis, are alone present. The diagnosis can be arrived at only by a process of exclusion, and then the question as to whether the lesion is right or left sided has still to be answered."

Under the term "frontal region" only that part of the brain is included which lies anterior to the ascending frontal convolution. Electrical stimulation of the anterior two-thirds or prefrontal region yields negative results as to motor changes, but its destruction has brought about ill-defined mental changes. In the posterior third or postfrontal region, stimulation experiments have demonstrated the presence of centers for turning the head and eyes to the opposite side and for some other ocular movements; and destruction of this region has produced paralysis of this movement and deviation of the head and eyes to the side of the lesion. These higher oculomotor centers lie in the hinder parts of the first and second frontal convolutions. Further, in the posterior end of the third frontal convolution of the left side lies the specialized motor center for speech, in the so-called Broca's convolution.

The general symptoms of intracranial tumor—headache, optic neuritis, and vomiting,—are usually present in cases of frontal tumor, the first two having some localizing value. The headache is as a rule frontal, but not infrequently occipital. It is often referred to the side of the tumor, especially when the growth is situated on or near the surface of the brain. Optic neuritis appears in almost every case first in the eye on the side of the growth, and reaches a more intense degree in this eye. This is so constant a

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(1) Lancet, Nov. 3, 1906.

feature as to be of real localizing value. If neuritis has been present for a long time the advent of secondary changes greatly diminishes its localizing value.

The mental symptoms associated with frontal tumor may be indirect from pressure or direct from immediate involvement of brain tissue. Undoubtedly the occurrence of mental symptoms in cases of frontal tumor has been greatly exaggerated; yet there is a large number of cases in which mental symptoms clearly preceded the onset of the more general and localizing symptoms. Hence it is very important to inquire of friends and relatives as to the character of the former mentality. The onset of mental symptoms is as a rule gradual, and usually precedes the gross signs of tumor. The most prominent and constant changes are inattention and inability to keep the mind fixed on the subject at issue, associated with a tendency to irrelevance and incoherence in conversation. In some instances a peculiar slowness in answering even the most simple questions is observed, though the answers may be entirely correct and intelligent. "A loss of memory more especially for recent events, is often a prominent feature; but perhaps most characteristic of the site of growth are those slight and indefinite mental changes and alterations of temperament which are difficult to analyze or describe. The patient is casual and irresponsible in word and deed, and may attempt jokes and make remarks which are quite irrelevant and often rude; this is apparently due to a faulty or too free association of ideas which is not held in check by a rational judgment and a normal power of concentration on the subject. In cases of long duration the psychic state may deteriorate to dementia or the patient may develop delusions and mania and in all cases any great increase in the intracranial tension gives rise to a stuporous or comatose condition. My personal experience is not sufficient to determine whether mental changes are more pronounced in lesions of the right or left frontal regions, but certainly, as far as tumors are concerned, my series of cases does not support Phelps's opinion that the occurrence of mental symptoms is associated only with left-sided lesions.

"In cases in which both frontal lobes are affected the psychic symptoms are more pronounced. Recovery of mental power after operation is greatest in those cases in which the growth is extracerebral and the frontal lobes only involved secondarily, as in cases of fibroma or endothelioma, while in cases in which the growth involves the brain substance directly as glioma the recovery is never so rapid or so complete.

"A second very characteristic feature of frontal tumors is the occurrence of various forms of fits and seizures. Epileptiform seizures seem to be especially frequent in frontal tumors. Such attacks were noted in 19 of the 22 cases of this series, and Müller states that out of 168 cases of frontal tumor more than one-third had epileptiform seizures either general or focal in character. These attacks may be classified into four chief varieties: (*a*) subjective sensations often described as "giddy feelings"; (*b*) petit mal attacks; (*c*) generalized epileptic seizures; and (*d*) fits commencing with a localized convulsion. The feelings of faintness or giddy attacks are not accompanied by loss of consciousness. The second class is distinguished from the first by a transitory loss of consciousness. They are identical with the petit mal attacks of idiopathic minor epilepsy. The third class consists of generalized convulsive seizures, similar to major idiopathic epilepsy. The fourth variety of seizure includes all the attacks with focal origin. The occurrence of fits with focal beginning reveals plainly the side of the tumor, and the absence of any sensory aura makes any question as to the post-Rolandic situation of the growth untenable.

"Symptoms which can be referred to affection of the cranial nerves are absent in a great number of cases of frontal tumor. When present they occur on the same side as the tumor and indicate that the growth is situated on or extends towards the base of the skull. Here a reservation must be made owing to the occurrence of cranial nerve palsies from indirect pressure due to the increased intracranial tension which results from cerebral neoplasm. The function which suffers most frequently from this cause is that of the sixth nerve which leads to convergent strabismus. Olfactory symptoms are very important when other causes, such as nasal disease, can be excluded; loss of smell, especially when unilateral, is of great significance.

"Paresis occurs only when the tumor impinges on the motor cortex or its efferent fibers and then contralateral hemiparesis of varying degrees may result. The most definite weakness is in many cases of the opposite side of the face which seems to be represented further anteriorly than are the limbs. Apart from such signs which are not strictly of frontal origin, there is one to which particular attention must be drawn as it has not been previously described. It is the occurrence of tremor in the limbs of the side homolateral to the tumor.

"This tremor is more constant and better marked in the arm than in the leg. In character the tremor is fine, rapid, and vibratory, and it may often be more easily felt than seen. It is absent during muscular rest and can be best demonstrated by making the patient extend both arms horizontally in front of him with the palms directed downwards and the fingers extended. This tremor may not be constantly present in any one case, but in all cases of my personal observation when the patient's condition has enabled it to be looked for it has been observed at one time or another. It occurs in cases in which there is no evidence of interference with the motor system, as well as in cases where such paresis exists. I have never met with similar tremor in association with tumors situated in other regions of the brain.

"Affection of sensation never results directly from a frontal lesion and only occurs after severe fits when the discharge has presumably passed across the motor centers to the post-Rolandic gyrus. The condition of the reflexes is of great importance. Tumors limited to the pre-frontal region do not as a rule cause any change in the deep reflexes, but when the growth impinges on the motor area the contralateral deep reflexes are increased. The superficial abdominal reflexes may be definitely affected in cases in which hemiparesis or other focal signs are absent. The first demonstrable sign is absence or diminution of the epigastric or abdominal reflexes on the side contralateral to the tumor, or it may be found that on repeated stimulation the contralateral reflexes are more easily exhausted than the homolateral. In all of my series of cases this diminution or loss of the superficial abdominal or epigastric reflexes on the contralateral side was observed.

The diminution or loss of these reflexes which occurs in acute abdominal conditions, such as appendicitis and enteric fever, is, as a rule, bilateral. We may conclude that the loss of the superficial abdominal reflexes on one side in any uncomplicated case of cerebral tumor is direct evidence of the tumor being situated in the opposite cerebral hemisphere.

"To summarize, in diagnosing a tumor of the frontal region we have to rely on (*a*) the presence of the general symptoms of intracranial growth; (*b*) the presence of mental symptoms; and (*c*) the absence of focal signs pertaining to other regions of the brain. For localization we must study the nature of any fits which may occur and be guided by the following signs. 1. Those homolateral to the tumor: (*a*) the earlier development and greater intensity of the optic neuritis; (*b*) the presence of a fine vibratory tremor in the extended limbs; (*c*) the presence of focal cranial nerve symptoms; and (*d*) the presence of local external signs. 2. Those contralateral to the tumor: (*a*) diminution or loss of the superficial abdominal or epigastric reflexes; (*b*) the presence of an extensor or indefinite plantar response with increase of the deep reflexes; and (*c*) hemiparesis. Though one or more of these signs may be absent it will be found that the majority of cases of tumor of the frontal lobe can be accurately diagnosed and localized, and this is the more important as there is in no other region of the brain, with perhaps the exception of the cerebellum, in which surgical interference can be advised with a greater prospect of success and with so little danger to the subsequent physical and mental condition of the patient."

Charles E. Beevor,[1] in the Lettsonian Lectures, discusses the diagnosis and localization of cerebral tumors. Tumors involving the cortex in front of the ascending frontal convolution, and comprising the superior, middle, and inferior frontal gyri may be classified as: 1. Tumors outside the brain. 2. Tumors growing in the cortex on the outer surface and situated in (*a*) the anterior half or (*b*) the posterior half of the three frontal convolutions. 3.

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(1) Lancet, Feb. 9, 23, and March 16, 1907.

Tumors growing within the white substance of the frontal lobe.

Of type one, tumors outside the brain, Beevor reports two characteristic cases. In the *first* case "the localization to the left frontal region was made by the following symptoms: The pain in the head was in the frontal region; the mental condition of stupidity and dulness, the attacks of coma, and the want of interest in the interval between attacks, all suggested the left frontal lobe; as did furthermore the incontinence of urine. In this case incontinence was associated with a depressed mental condition, and the interesting point is that on the very day of the operation, when the pressure was relieved, this symptom passed away. The "other symptoms which helped to localize the tumor on the left frontal region were: 1. The impairment of smell on the left side as compared with the right. 2. The slight paresis of the right arm and leg. 3. The increase of the deep reflexes (wrist tap, knee-jerk) in the arm and leg on the right side as compared with the left and diminution of the superficial reflexes, abdominal and epigastic, on the right side. The plantar reflex gave a doubtful extension of the right big toe as compared with the definite flexion of the left toe. The paresis of the right side with increase of the deep reflexes and diminution of the superficial reflexes are the common symptoms of a slight hemiplegia, and in this case would doubtless be caused by the general compression by the tumor of the pyramidal tracts of the left side and thus pointing to a lesion of the left hemisphere. 4. Fine rapid tremor of the left hand, when both arms were extended."

Of type two, tumors involving the outer surface of the frontal lobe, Beevor reports five cases, four of which were under the care of Sir William Gowers. "In all these five cases the tumor involved chiefly the second or middle frontal convolution. In three cases where the tumor was near to the ascending frontal convolution the fits affected first the fingers, or the conjugate movement of the eyes, or the angle of the mouth, while in two cases where the tumors were in the anterior part of the second frontal gyrus there were attacks of giddiness or fainting without convulsions, more like petit mal. It would appear that the presence of fits, either faints or attacks beginning with spasm of conjugate movement of the eyes to, let us say, the right, or of the hand or face followed by weakness but not by permanent anesthesia and with increase of the deep and diminution of the superficial reflexes; of mental symptoms; and of fine tremor on the left side, opposite to that affected by the fits and the weakness, are the diagnostic signs of tumor of the outer surface of the cortex of the left frontal lobe anterior to the ascending frontal convolution."

Of the third group of frontal tumors, those involving the internal white matter of the frontal lobe, Beevor presents notes on ten cases. The symptoms differed according to the degree to which the tumor extended posteriorly and whether the internal capsule was compressed, for in none of the ten cases was it actually involved by the growth. In six of the cases there were severe headache, vomiting (except one case), double optic neuritis, mental obtuseness, want of memory and attention, and with incontinence of urine in most of the cases, but no difference in the sense of smell on the two sides; a gradual hemiplegia without any anesthesia, but with increase of the deep reflexes, diminution of the superficial reflexes with extensor plantar response on the paralyzed side. In three of these cases there was a fine rapid tremor of the hand on the same side as the tumor. The headache is often frontal but it may be on both sides. Smell is not affected on one side as it is in extramedullary tumor.

The deep and superficial reflexes may not be altered though they are usually diminished. Mental dulness is often a prominent symptom and also involuntary micturition, but they do not help us as to which frontal lobe is involved. When the tumor extends backward so as to compress the motor fibers of the corona radiata or internal capsule, gradual weakness of the face and limbs on the side opposite to the tumor comes on with increased deep reflexes and diminished abdominal and epigastric reflexes with extensor plantar reflex, but without any affection of sensation. As soon as these symptoms appear they point to which side of the brain is affected and this, taken with the mental symptoms and the involuntary micturition and the fine tremor on the same side as the tumor, may enable a diagnosis of a frontal tumor to be made and also of the hemisphere in which it is situated.

Tumors of the temporal lobe are described by Charles E. Beevor[1] and ten cases are partially reported as illustrative of the symptomatology. "The position of tumors of the temporal lobe is very difficult to diagnose, because the area is very large and only two centers—viz., those for taste and smell and those for hearing—have been localized. We have to rely on fits having a special character to make a diagnosis of tumors of this part. The fits were first called attention to by Dr. Hughlings Jackson in 1876, who describes them as 'intellectual auræ or intellectual warnings of epileptic seizure.' "

In three of the ten cases the anterior part of the temporal lobe was involved, but not the gyrus uncinatus. "In one of these cases the patient had fits with the aura of a horrible smell; in the other two cases the sense of smell on both sides was bad, but this may have been due to excessive intracranial pressure. Hearing in the opposite ear was lost in one case and diminished in another, and in both cases the anterior part of the superior temporal was involved. Of the other cases, in one with a tumor of the gyrus uncinatus there were fits of the 'dreamy state' with a bad taste lasting all day; and in a case of tumor of the anterior part of the temporal lobe, including the gyrus uncinatus, the patient had the feeling of the 'taste of chalk' occurring spasmodically.

"In all the above cases there was always great difficulty in localizing in which hemisphere the tumor was, especially in tumors about the uncinate region, but in nearly all the cases there was weakness of the opposite face or limbs, or increase of the deep reflexes, or diminution of the superficial reflexes, with extensor plantar reflex on the side opposite to that of the lesion due to secondary pressure of the growth on the internal capsule, which gave some help to the diagnosis."

Tumors of the Supramarginal and Angular Gyri. These tumors are discussed by Charles E. Beevor,[2] who believes that these gyri are probably inexcitable to electric stimuli in man. Five cases are described in detail, two being in

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(1) Lancet, Feb. 23, 1907.
(2) Lancet, March 16, 1907.

the left hemisphere and three in the right. The general symptoms of tumor were either absent or very late in developing. Headache was present throughout in the first case and was severe in the third case, but in the other cases it was not severe and was absent till the end. Vomiting did not occur at all in three of the cases and only at the end in two cases. Optic neuritis was marked in the first case, it was absent in the third case, and in the other cases it was not observed till very late in the course of the disease. In all cases there were either general convulsions without any warning, faints like petit mal, or Jacksonian attacks without loss of consciousness. These last fits were always preceded by a sensory aura. In one case hemiplegia occurred early in the case, but in all the others there was only slight weakness of one side and this weakness occurred late either with normal reflexes or with the increase of the deep and diminution of the superficial on that side. Sensation in two cases was not permanently lost, although in both cases the patient had attacks beginning with a sensory aura, but in the other three cases there was hemianesthesia and in two cases hemianalgesia. Hemianopia of the fields on the side opposite to the tumor was present in every case except two, and in one of these the field of the opposite eye was concentrically contracted. The sense of smell was deficient on the same side as the tumor in two cases. The most important symptom was the affection of speech. In the three last cases when the tumor was on the *right* hemisphere speech was not affected; in the first two cases affecting the *left* hemisphere there was a definite amount of word blindness.

In the foregoing account the fits, which begin with a sensory aura, are probably due to pressure on the ascending parietal convolution with consequent irritation of that part. Paralysis of motion and sensation does not occur unless the motor or sensory fibers of the corona radiata or internal capsule are pressed upon or involved by the tumor. The permanent hemianopia would probably be due to the optic radiations being invaded, while the concentric limitation observed in one eye would be due to a lesion of the angular gyrus. The particular affection of speech called "word blindness," is considered to be situated in the left angular gyrus, though in the first of these cases the tumor was removed from the supramarginal and probably compressed the angular gyrus. Therefore we may say that with tumors in the parietal lobe posterior to the ascending parietal convolution the general symptoms tend to be absent or to appear late in the disease; the fits are general or begin with a sensory aura; paralysis of the side opposite to the tumor with the accompanying alteration of the reflexes is absent, slight, or occurs late, unless the tumor extends deeply, as in the last case, and involves the internal capsule; hemianesthesia and hemianopia on the opposite side occur frequently, and if they are permanent it would show that the sensory fibers and the optic radiations were destroyed. If the tumor be in the *left* parietal region word blindness, either transient or permanent, is present.

Tumors of the Optic Thalamus. Charles E. Beevor[1] describes these tumors and gives notes on three cases. The optic thalamus is a part of the brain of which the function is less known than that of other parts, such as the cortex. The chief symptoms of its disease are that voluntary movements are slightly affected unless the internal capsule is involved, but involuntary movements and ataxia of the opposite limbs occur; the tendon or deep reflexes are slightly increased or are normal; the superficial reflexes are normal or absent, but the extensor plantar reflex (Babinski's sign) was not obtained in spite of the pyramidal degeneration which was present in some of the three cases on which this summary rests.

Sensation was affected both objectively and subjectively; in the former all forms of sensation are involved but not absolutely, and the loss is more marked at the periphery. Muscular sense and stereognosis are completely lost. Subjectively there are severe pains in the limbs and hyperesthesia, which is sometimes so severe as to prevent the use of the limb. Hemianopia occurs if the postero-inferior part of the thalamus be involved. The other special senses are not affected. The internal capsule lies so close to the optic thalamus that it is very difficult in the case of tumor not to have the internal capsule involved or pressed on.

"A history, therefore, of a slow progressive hemiplegia

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(1) **Lancet, March 16, 1907.**

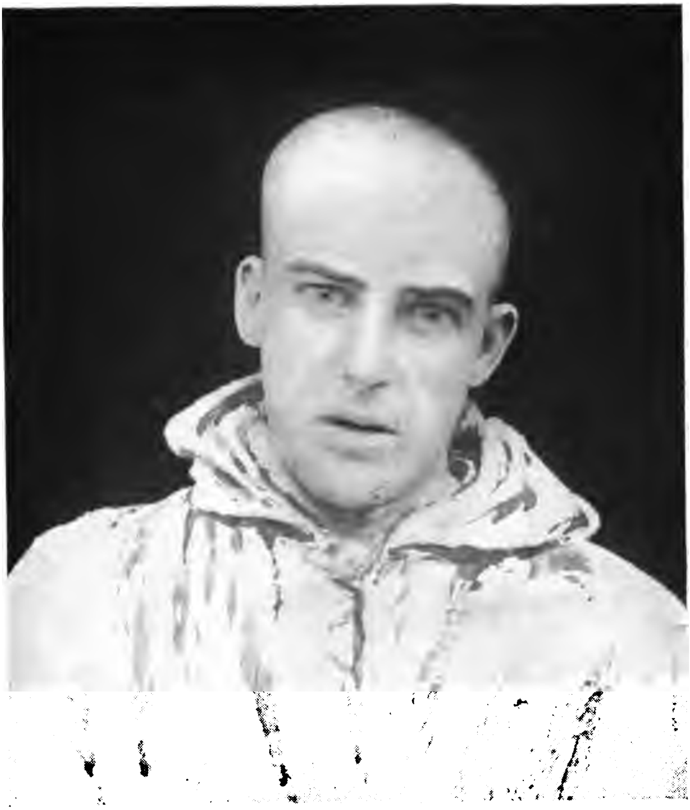

PLATE I.

Showing the protrusion subsequent to the development of a hernia cerebri following a decompressive operation, in which, after exploration, the dural wound was closed. (Page 117.)

with hemianesthesia and hemianopia all on the same side, with fits of a general nature but without any localizing symptoms, such as numbness in one hand followed by epileptiform attacks, as are found to exist in tumors of the parietal lobe, would make a diagnosis of a tumor about the optic thalamus and the posterior limb of the internal capsule probable. In both the cases where the internal capsule was involved the plantar reflexes were extensor in character and not flexor as described by Déjerine and Roussy, and in none of the cases were there severe pains in the limbs. In one case ataxia of the paralyzed side was observed."

Tumors of the Corpus Callosum. At the Congress of Alienists and Neurologists held at Lille in August, 1906, Raymond[1] reported two cases of tumors of the corpus callosum with subsequent postmortem examinations. [It is not necessary to recount these histories in detail, but Raymond's remarks concerning them are of value. They agree in the main (but not wholly) with Putnam's conclusions previously recorded in the PRACTICAL MEDICINE SERIES, 1902, Vol. X.—ED.] Raymond attributes to the involvement of the corpus callosum practically only one symptom, namely, mental impairment. "The intellectual beclouding, the demential disturbances which certain authors have described in tumors of the corpus callosum, are late symptoms due to the spread of the tumor to neighboring regions or to diffuse cerebral alterations such as those resulting from compression, intoxication, or vascular lesions.

There remains, as characteristic, a special mental syndrome: it consists of disturbances of character, of oddities in manners and acts, gaps in memory, a lack of correlation in the ideas, associated notwithstanding with an apparent preservation of the intelligence which may deceive those about him. The localization of a tumor in the corpus callosum may be suggested not only by such a mental syndrome, but also by the site of the headache, but only by the very earliest headache. Other symptoms recorded are doubtless neighborhood symptoms, the tumor being rarely accurately limited to the corpus callosum." On the mental

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(1) Progrès Méd., Aug. 25, 1906.

syndrome described Raymond accurately located the tumor in the second of his two cases reported. Raymond is unwilling to draw any conclusion as to the physiology of the corpus callosum. The occipito-frontal bundle was destroyed in both cases, and possibly not even this mental syndrome can be ascribed solely to the corpus callosum alone.

Tumors of the Hypophysis Cerebri. Harvey Cushing[1] calls attention to the fact that tumors affecting the hypophysis cerebri are not only apt to be associated with optic atrophy, but they are also associated with sexual infantilism, whether occurring in male or female. He reports two illustrative cases. The first case seems to show that a slow-going, congenital, intracranial tumor, mesially placed and so situated as to compress the hypophysis cerebri, can for years give no symptoms (barring an occasional headache) other than a retardation of sexual development accompanied by some obscure nutritional disturbances. The rather acute onset in this case, of all intracranial symptoms, with rapid bilateral choked disk, was in all probability due to the final production of an internal hydrocephalus consequent on the projection of the tumor into the third ventricle. The only objective evidence of ocular disturbance, before choking of the disks became apparent, lay in the narrowing of the visual fields and some pallor of the nerve-head.

Treatment. William M. Leszynsky[2] discusses the treatment of brain tumors. When syphilis is known or suspected, mercury and iodids may prove of value, although they are often not effective even when a syphilitic gumma is present. For headache, some of the coal-tar products are available, or ergot or morphin may be given subcutaneously. At times free catharsis helps. Lumbar puncture is of doubtful value and often makes matters worse rather than better. Operation should be resorted to as soon as a positive diagnosis is made, and after the condition has proved unamenable to medical treatment. If the tumor has been accurately localized and is accessible, its removal should be undertaken if possible. If it cannot be removed, the relief of intracranial pressure will tend to

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(1) Jour. Nerv. and Ment. Dis., November, 1906.
(2) Med. Record, July 13, 1907.

prevent blindness, and a decompressive operation should be performed over the right temporal region; for in this location no harm can result through the formation of a cerebral hernia. In a left-handed individual the operation is performed on the opposite side. "In doubtful cases, particularly when a cerebellar growth is suspected and when vision is not at all impaired, we should not be too hasty in recommending surgical intervention. We must admit that the greater number of brain tumors are inaccessible to operation, and, although occasional brilliant results are achieved, the surgical treatment of brain tumors is still a difficult problem."

Cerebral Decompression. William G. Spiller and Charles H. Frazier[1] discuss at length the subject of cerebral decompression, basing their remarks largely on an exhaustive review of the literature, and personal operations on fourteen cases. A careful study of their own cases and of those reported by others has led Spiller and Frazier to favor decidedly the palliative operation. The choked disk, headache, vertigo, nausea, vomiting, and to some extent the convulsions are favorably influenced by this treatment, and the relief is often permanent. Yet, it is a mistake to regard palliative operations as a substitute for radical operations. The tumor should be removed whenever this is possible, and palliative measures are to be considered only when the tumor cannot be located, or is too large for removal, or possibly is a glioma. Spiller does not agree with Horsley that a palliative operation can lead to the atrophy of the tumor, nor does he believe that it favors the growth of the tumor by release of pressure. Spiller thus summarizes his views:

1. Palliative operations should be performed early in every case in which symptoms of brain tumor are pronounced and before optic neuritis has advanced far, especially where syphilis is improbable, or antisyphilitic treatment has been employed. 2. Partial removal of a tumor, especially of a glioma, is a questionable procedure. 3. Palliative operation does not cause atrophy of a brain tumor and probably does not arrest its growth; on the other hand, it probably does not hasten its growth. 4. Palliative ope-

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(1) Jour. Am. Med. Assoc., Sept. 1, 8, 15 and 22, 1906.

ration is not to take the place of a radical operation when the latter can be performed without great risk to the patient. 5. In some cases the symptoms of brain tumor disappear almost entirely for a long time or permanently after a palliative operation. This result is obtained either by relief of intracranial pressure or by removal of some lesion (meningitis serosa, etc.) other than brain tumor, and yet causing the symptoms of tumor."

Charles H. Frazier believes that the question as to what percentage of cerebral tumors may be removed will be determined only by statistics gathered on the operating table. As to decompression operations, there are two classes of cases which may require surgical interference; one in which the tumor cannot be removed in its entirety, and the other in which the tumor cannot be localized; and yet the possible loss of vision, the intense headache and distressing vomiting almost demand the immediate adoption of some measure of relief. Tumors of the cerebrum in which localization is impossible are rather the exception, although cerebellar lesions not infrequently cannot be localized. Failing to find the cerebellar tumor at operation, Frazier has deliberately removed from one-quarter to one-third of the cerebellar hemisphere, and has attributed the relief of pressure, in part at least, to this.

First, as to those cases in which the tumor cannot be localized, there are two points to be made. When the situation of the tumor is unknown, the operator is free to select any one of four areas: the frontal, parietal, occipital, or temporal. Cosmetic reasons preclude operation on the frontal and occipital regions. Moreover it is better to select a silent area like the right temporal, since no aftereffects will follow.

Secondly as to cases in which the tumor is localized, but its nature and size are unknown, it cannot be told in advance whether it is operable. In every such instance the surgeon should begin with the intention, first, of exposing the tumor. Failing to find the growth or finding an inoperable one, a decompressive operation is indicated. He must then determine whether he will remove the bone of the flap, wherever this may be, or close the flap and reopen over the area of choice—the right temporal region. When, as is true in most cases, the tumor lies in the postfrontal (motor), or parietal region, the bone over the temporal area may be removed through the same opening, thus avoiding another incision.

The most important question, however, is as to whether the dura shall be incised or removed. Frazier does not believe that benefit follows the mere escape of a little cerebrospinal fluid through a temporary opening of the dura, and he furthermore does not believe that drainage through a permanent opening will afford any relief. He does believe, that the dura should be left intact. The claim that the dura is not elastic enough to stretch when subjected to pressure is disproved by Frazier's cases. Plate I shows the protrusion in the right parietal region after a decompressive operation in which, after exploration, the dural wound was closed. Fig. 4 shows the rapid development of hernia cerebri in diagrammatic form. Clearly in this latter case, the dura stretched, and stretched rapidly.

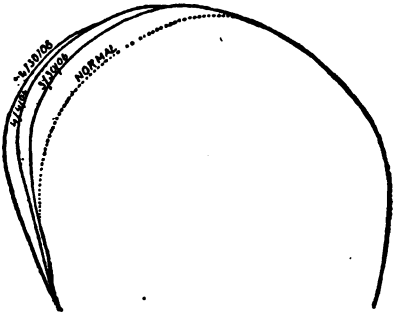

Fig. 4.—Showing the rapid development of a hernia cerebri in the decompressive operation. After the operation the dural wound was closed. The dotted line represents the normal contour on the affected side. The other lines represent the contour of the head: (1) March 30, 1906, one week after the operation, (2) April 4, 1906, twelve days after the operation, (3) April 30, 1906, five weeks after the operation. Same case as Plate I.

"Inasmuch as the brain may thus herniate through an opening in the skull when the dura is left intact, there is really no necessity for removing more than the overlying bone. If the dura is disturbed at all the operator should be content with making a crucial or radiating incision in it, and in many cases it may be advisable to reserve this step of the operation until indications arise for further intervention."

As to the mortality from the operation it may be stated that every one of the eleven cases here reported by Frazier and the one case operated on by Martin at the University Hospital, recovered. "When one is debating, therefore, as to the advisability in a given case of performing a radical or palliative operation, consideration should be given to the fact that there is at least no immediate risk as to life from the decompressive operation. In the palliative measure we have an operation which almost inevitably relieves the sources of discomfort and suffering, not only without endangering but frequently prolonging life; in the radical measure we are undertaking an operation which has a comparatively high mortality and is only successful in a small percentage of cases in finding and removing the tumor."

"The decompressive operation offers to the patient relief from the three cardinal symptoms of cerebral tumor, headache, nausea and vomiting, restores vision, and, if the lesion is in the cerebellar fossa, relieves ataxia and vertigo. . . . . One of the strongest arguments in favor of the palliative operation is the opportunity to save or restore the patient's eyesight. Without exception the choked disks subsided in every instance, but unfortunately in at least three of our cases the patients remained totally and hopelessly blind because the optic neuritis was of so long duration that the nerve had undergone atrophy. The subsidence of the choked disk takes place as rapidly when the dura is left intact as when it was incised. . . . The prospects of preserving the eyesight alone would justify the operation, but no less so the opportunity of relieving headache. Our results with respect to headache have been uniformly good. There were two or three cases in which the headaches did not entirely cease, but in these there were intervals of relief and the headaches were less severe. As to the duration of the period of relief the longest in our series is between two and one-half and three years. What has been said of the beneficial effect of the operation on the choked disk, even when the dura was left intact, may be repeated in dealing with headache. In conclusion we may say that there is no form of treatment, either medical or surgical, which, in the presence of an incurable lesion, causing sometimes great discomfort, sometimes intense suffering, so uniformly affords so great and enduring a measure of relief."

Concerning operation for brain tumor, T. H. Weisenburg[1] may be quoted as follows: "Although in the majority of instances, the removal of a brain tumor is not accomplished, this fact should not deter surgeons from operating, for an exploratory operation is at least as much indicated on the skull as on the abdomen. Such an operation has a double advantage in that it relieves intracranial pressure, thereby not only ameliorating the immediate symptoms, but also relieving headache and often preventing blindness by lessening the severity of the choked disks. In fact, too much emphasis cannot be placed on the advantages of an early exploratory, or so called palliative operation.

OPTIC THALAMUS.

Optic Thalamus Syndrome. Déjerine[2] describes the optic thalamus syndrome, illustrating his remarks by a patient of 50 years of age, who in January, 1901, after a violent assault, suddenly was seized with a right-sided hemiplegia. She did not lose consciousness or power of speech. The paralyzed side quickly became hemianesthetic, and the next day showed a bilateral homonymous hemianopia. After a few months the hemiplegia wholly disappeared, but hemianesthesia persisted. At the same time severe pains appeared in the right side of the body, of a lancinating type, almost intolerable. In this state she remained until last year, when she was again examined. Strength was almost equal on the two sides. In repose she showed certain choreic-athetoid movements, disappearing during sleep. The tendon reflexes were normal and Babinski's toe-sign was wanting. There was a slight diminution of sensibility to pain, temperature, and touch on the right side, and sensibility to pressure was much diminished. Muscle-sense was gone; she could not tell in what positions her joints were placed, if blindfolded. The stereognostic sense is abolished. There was a slight ataxia,

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(1) New York Med. Jour., Oct. 3, 1906.
(2) Gez. des Hôpit., Jan. 25, 1907.

Summarized there was hemianesthesia of the cerebral type with hemihyperesthesia, without hemiplegia, or exaggeration of the reflexes; and also choreic-athetoid movements, hemiataxia, and especially very intense pains in the afflicted side. Such is the thalamic syndrome.

CEREBELLUM.

Tumor of the Middle Lobe. In *Brain*[1] appears the report of a case of tumor of the middle lobe of the cerebellum, showing cerebellar paralysis with rigidity, and occasional tetanus-like seizure, originally made by J. Hughlings Jackson, in 1871, in the hospital reports of the *British Medical Journal,* and now reprinted in the number of *Brain* dedicated to him as a tribute of respect and affection in the fiftieth year of his medical practice by the neurologic society of the United Kingdom. The diagnosis then made of tumor of the middle lobe of the cerebellum pressing on the veins of Galen and the straight sinus was made "with confidence" by Hughlings Jackson, and was confirmed at the postmortem examination. The symptoms of the case were analyzed with great skill and the proper deductions drawn. In the same issue of *Brain* is the reprint of a second case somewhat like the first, originally published in 1872 and also properly diagnosed, and situated in the same location. These cases, besides being an excellent tribute to the analytical powers of Hughlings Jackson are an excellent illustration of the fact that our knowledge of cerebellar disease and function has after all very slowly increased.

Functions of the Cerebellum. Victor Horsley[2] sets forth Hughlings Jackson's views on the functions of the cerebellum. In 1877 Jackson wrote: "The cerebellum is the center for continuous movements, and the cerebrum for changing movements. Borrowing medical terms to describe normal physiologic conditions, the former (that is, *cerebellar*) are tonic, the latter (that is, *cerebral*) are clonic." This has been proved to be true by François-Frank and Pitres, and confirmed by Victor Horsley and

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(1) March, 1907, Part iv, 1906, p. 425.

(2) Brain, March, 1907, No. 116; British Med. Jour., April 6, 1907.

others. Their well known work consisted in the demonstration that whereas the muscle-contraction phenomena evoked by excitation of the cortex cerebri were essentially clonic in character, those obtained by stimulating the pyramidal fibers of the corona radiata conducting to the lowest level centers were tonic. It is impossible to summarize the whole of Horsley's valuable paper. It will suffice to give his main conclusions.

First, the cortex cerebelli is inexcitable when compared to the cortex cerebri of the gyrus centralis anterior (ascending frontal gyrus). The immediate inference from this result is that the cortex cerebelli is not a motor center or series of such centers, but is an afferent receptive center from which axones pass to the ventrally-placed efferent nuclei.

Second. The intrinsic and the paracerebellar nuclei are the efferent mechanisms of the cerebellum. When stimulated electrically these nuclei cause muscular contractions. As yet Horsley's researches are not complete as to the topographic representation in the cerebellar and paracerebellar nuclei, but provisionally it may be asserted that the essential motor representation of the parts of the body in these nuclei are in the case of the intrinsic nuclei the movements of the eyes and head, and in the paracerebellar nuclei the movements of the trunk and limbs. "The character of the motor effects obtained is very striking and precisely of the kind originally ascribed by Jackson to it, namely, tonus and hypertonus. The movement even of the eyeballs is a steady swing over, which increases in contractile force as the excitation continues and without any clonic intermission of the motion or any development of any after-effect when the stimulation ceases. This being understood, the next questions respecting the character of movements derived from the efferent cerebellar mechanism are: (*a*) their relation to the middle plane of the body; (*b*) differentiation between flexion and extension.

"(*a*) *Relation of the Cerebellar Influx to the Mid-plane.*—We find that when the stimulus is unilaterally applied to the intrinsic cerebellar nuclei, the effect is homolateral, contrasting with the typical cerebral contralateral phenomena. A demonstrative example and proof of this principle is afforded by the result of our direct excitation of the upper part of the dentate nucleus alone, which we find evokes deviation of the eye of the same side, followed by conjugate deviation of both the eyes and head from the middle line to the same side. So, too, excitation of the basal region of the dentate nucleus and the upper divisions of the paracerebellar nuclei of one side evokes powerful bicipital flexion of the homolateral elbow.

"(*b*) *Relation of Flexion to Extension.*—As regards the second point, Clarke and myself find, that while excitation of the basal region of the dentate nucleus with a maximal stimulus will produce, in addition to the homolateral conjugate deviation of the eyes and head, flexion of the homolateral elbow, a deeper excitation, that is, of the paracerebellar nuclear region itself, produces widespread and additional effects, namely, extension of the contralateral elbow, hyperextension of the neck and trunk, with powerful extension of the lower limbs. While, therefore, the forelimb is flexed at the elbow the hind limb is extended with the trunk."

Symptomatology. The functions of the cerebellum and the symptoms of its disease are described at length by Charles L. Dana[1] and six illustrative cases are recorded: The essential function of the cerebellum is equilibration. Indirectly it helps in coördinating all muscular movements, from those of the limbs and trunk to those of the eyes. Direct cerebellar symptoms are vertigo, with irregular ataxic and forced movements, and loss of equilibrium. The sense of vertigo is one of the commonest of disagreeable sensations, because the cerebello-spinal mechanism has so many factors in its makeup. Thus the nerves from the labyrinth, the visual nerves, the cutaneous and muscular sensations all have much to do with equilibration. Another direct cerebellar symptom is atonia. The cerebellum gives a certain degree of "tonus" to the muscles, so that they are always ready to respond to the proper stimulus. When the cerebellum is injured, therefore, there may occur loss of deep reflexes and muscular atony, as in locomotor ataxia, though less marked.

The cerebellum also has the function of regulating movements so that flexors and extensors respond in proper bal-

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(1) New York Med. Jour., Oct. 6, 1906.

ance, and so that each segment of the limb follows in its movements in proper order. This is called synergy. "When the cerebellar 'tonus' and synergy of the muscles are lost the muscular effort is attended with labor and weakness, and we have a symptom known as parasthenia. This is interpreted by the patient as a general weakness and easy fatigability, and such patients have attacks or periods of exhaustion which appear like conditions of neurasthenia. The lack of power to properly tone and coördinate muscles leads also to rhythmic contractions or tremors of a coarse intentional type. It shows itself also in lateral oscillatory movements of the eyes and head. Further, the disorder of this mechanism leads to a constant feeling of wrong equilibrium, so that the body is held in a peculiar constrained position, as to the head or neck or the trunk. A common and characteristic symptom of cerebellar tumor, for example, is a wry neck position of the head, while in cerebellar lesions due to hemorrhage or sclerosis, the head is often carried in a stiff, rigid way as if the patient was balancing something on the vertex."

Direct Symptoms. The direct effects of lesions of the cerebellum and its afferent and efferent tracts then consist of: 1, Ataxic and reeling gait; 2, forced movements; 3, constrained attitudes; 4, tremors, nystagmus and head oscillations; 5, disorders of segmental peripheral movements of the hands or "asynergy"; 6, vertigo and its accompanying sensations; and 7, sense of weakness and myasthenia.

Indirect Symptoms. When a disease like a tumor or a hemorrhage occurs, there is compression of the cerebellum between the base of the skull and the tentorium; the aqueduct of Sylvius is stopped up, fluid accumulates in the ventricles, the nuclei of the cranial nerves on the floor of the medulla are pressed on, and a series of secondary symptoms follow. Though secondary, they are often early, important, and serious. "They consist of headache, vomiting, optic neuritis, and in the latter stages disordered breathing and pulse, and involvement of the oculomotor and other cranial nerves, and retraction of the head. These, however, are all pressure symptoms and not directly cerebellar."

"In experiments on animals in which one-half the organ is cut away, or the peduncles are cut, other more striking features are observed. The animal has retraction of the head and opisthotonos, or the trunk is twisted to one side. The limbs on one side are adducted, on the other abducted, there is forcible extension or flexion of the limb, and the animal lies in an awkward, sprawling position. With this there may be clonic movements of the head and eyes, forced rotation of the body, and violent tremors.

"These symptoms are rarely seen in ordinary chronic cerebellar disease, but they do occur as an epiphenomenon at the onset of some acute diseases, such as hemorrhages, and in the course of some chronic diseases, such as tumors. In these cases the patient suddenly has a sense of pain or of rushing of blood to the head, with vertigo and roaring noises in the ears, stiffening and irregular tonic spasm of the limbs, the patient lying in a sprawling attitude. Or the seizure may consist simply of a sudden accession of pain as if the head were struck by a blow, followed by a pitching forward or sinking in a heap to the floor.

"I have described these *cerebellar seizures* as occurring in three groups of diseases: 1, Tumors, especially those of the cerebellar pontine angle; 2, hemorrhage of the cerebellum directly, and after the clot is absorbed leaving an irritating cicatrix; 3, in cerebellar abscess; 4, in primary defects of the cerebellum; 5, in neuropathic constitutions as epileptic equivalents.

"Thus we have three groups of cerebellar symptoms: 1, Those due to direct injury or irritation of the cerebellum; 2, those due to increased intracranial pressure, and thus causing general cerebral or local pons-medullary symptoms; and 3, those due to sudden irritations or vascular changes leading to seizures."

There follows the report of four cases of cerebellar hemorrhages or softening, one of them showing a slightly ataxic gait, a somewhat constrained position of the head, slight nystagmus, tremor, vomiting, and severe vertigo, with at times some general tinnitus and muscular weakness. The syndrome in another case was: 1, An attack of severe vertigo, objective and subjective; 2, a second attack with vomiting, projectile in character; 3, headache with mental confusion and apprehension at times; 4, mildly abortive attacks of vertigo, consisting of a sensation like a flash coming up into his head, and a feeling as if he would drop; 5, lateral oscillation of the head when turned to right or left; 6, no static ataxia, but a tendency to fall backward with his eyes closed; 7, some unsteadiness of gait; 8, attacks of excessive weakness; 9, no disturbance of the eighth nerve.

"*The syndrome of the cerebellar hemorrhage,* then, consists of headache, vomiting, and frequently with a seizure in which there is faintness and sometimes loss of consciousness, and sometimes tonic and clonic movements of the limbs. This is followed by a residuum of symptoms, which consist of headache or uncomfortable sensations in the region of the occiput, persistent vertigo, mainly subjective in character, increased on moving, and relieved by the recumbent posture; the peculiar attitude or constrained position of the head, some degree of ataxia, or a reeling gait, nystagmus, with oscillations, quicker in one direction than in the other, a general easy exhaustibility of muscular force, and sometimes spells or periods of muscular weakness, sometimes, also, with tremor of the hands, face, and tongue. In some cases the course of the residual symptoms is interrupted by attacks of severe vertigo, and even by attacks characterized by muscular spasms or cerebellar seizures. There may or may not be with these a good deal of tinnitus, and sometimes attacks of vomiting. The dominant symptom, however, in all of these cases is a vertigo, and feeling of inability to do the work, because all muscular exertions are accompanied with a sense of uncertainty and attitude. The condition naturally suggests somewhat that of tumor, but there are lacking the optic neuritis, vomiting, headache, and rapidly progressive character."

Symptoms of Functional Cerebellar Disease (Cerebellar Neurasthenia). There are cases in which the patient seems to inherit a defective cerebellum. These patients are neurasthenic, or parasthenic. They never are quick or agile on their feet, never able to dance well, or at all; they are easily tired, and have periods of special exhaustion and drowsiness. They are great sleepers. They have to walk carefully and hold their heads in a constrained position. They cannot move quickly without vertigo. They get sea sick and car sick very easily, they have sometimes an oscillatory spasm of the head, especially when tired. They may have some nystagmus. The handwriting is cramped and peculiar. They have attacks of severe vertigo, resembling labyrinthine disease, and these attacks may even end in unconsciousness and be accompanied with rigid spasms. In the early stages they often pass as neurasthenics.

Dana then considers each of the cardinal cerebellar symptoms by itself.

FIRST. Most important and frequent is *vertigo.* It is subjective, and often not so much a real vertigo as a sense of insecurity and fear of falling. The patient therefore seeks at once a place where he can lie down or brace himself. The vertigo is particularly apt to disappear when he is recumbent, and to reappear when he rises or stands. It is accompanied with feelings of weakness and exhaustion—an acute myasthenia. There is not apt to be nausea except when the vertigo is very intense, when the patients often fall to the ground and lose consciousness.

SECONDLY. The *nystagmus* differs from that due to peripheral and nuclear changes of the oculomotor nerves in that the movements are slower to one side than to the other, and that the quicker movements are towards the side of the lesion. The nystagmus is usually not persistent, but is brought on by turning the eyes to the side or up and down. Commonly it is a lateral nystagmus. Oscillatory movements of the head, "lateral head nystagmus," may be associated in these cerebellar lesions.

THIRDLY. The *constrained attitudes* often suggest the position of a person with a wry neck. [See PRACTICAL MEDICAL SERIES, Vol. X, 1904, p. 162.] The head is turned to one side and the chin a little elevated, and the body is sometimes curved away from the side toward which the head is turned. In some cases, the head is held fixedly, and the trunk is curved laterally, so that the patient stands as if he had a pain in the side. Plate II shows this special sort of lateral curvature, while Plate III illustrates the attitude in cerebellar disease.

FOURTHLY. The *muscular weakness,* or parasthenia, is manifested by easy fatigability, and frequently follows an attack of comparatively mild vertigo or cerebellar vomiting. The weakness ensues because the effort of working muscles that are not well coördinated is generally great.

FIFTHLY. The *gait* is reeling in serious cases, but not in those with smaller lesions. In the latter we find only a careful gait, with short steps and a wide base, inclining to one side or the other. Sometimes there is a kind of festination or pitching forward, such as is seen in paralysis agitans. While the cerebellar patient walks unsteadily, he can generally stand with closed eyes; if he cannot do so, he pitches backwards, forwards, or sideways, instead of swaying as does a tabetic. When the patient is recumbent he can move his limbs and head freely and purposely.

Cerebellar Peduncle. A. Porot[1] reports a case of isolated destruction of the superior cerebellar peduncle by a hemorrhage. The patient showed vertiginous attacks with vomiting, transitory diplopia, a slight retardation of the pulse (60), dysarthria, disturbances of movement, of equilbrium, and of coördination. Speech was slow and a little jerky. The patient walked with the legs far apart, and in the manner indicative of a cerebellar lesion; the motor troubles were chiefly on the right side, and clearly so. On the right side there was also asynergy, and ataxia in the right arm. Diadokokinesia was present in the same arm. At the postmortem examination a small hemorrhagic focus was found limited to the right cerebellar peduncle. The case permits of the following deductions:

(1) Lesions of the superior cerebellar peduncles give rise to the symptom complex of cerebellar lesions in general with vertigo, vomiting, and diplopia, and hemiataxia and hemiasynergy on the side of the lesion. (2) Such lesions give rise to *direct* symptoms, that is on the same side as the lesion, despite the statements to the contrary on the part of some. (3) Such lesions emphasize the great value of what Thomas says is characteristic of the cerebellar syndrome: the contrast between the disturbances of equilibration and movements, and the apparent integrity of muscle strength and of sensation in general. (4) Such lesions also emphasize the symptoms of asynergy and of diadokokinesia described by Babinski, and show that they may be unilateral. (5) Finally it is to be remarked that certain symptoms, such as hemiataxia and hemiasynergy, maintained a certain fixity of intensity throughout the course of the case, whereas the disturbances of equilibra-

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(1) Rev. Neurolog., Dec. 15, 1907.

tion and the vertigo improved up to the time of death, which ensued from tuberculosis.

TUMORS OF THE CEREBELLUM.

Symptomatology. Cerebellar tumors are discussed as to symptoms and diagnosis by Charles E. Beevor[1]. These tumors may be classified as: (1) Extra-cerebellar tumors, and (2) intra-cerebellar tumors, divided into (a) tumors of the lateral hemispheres, and (b) tumors of the vermis. There are the cardinal symptoms of tumor in general, headache, vomiting,. and double optic neuritis; and the following localizing symptoms: "Vertigo, pressure on the cranial nerves (auditory, facial, trigeminal, sixth nerve), motor symptoms on the side of the lesion, such as weak conjugate movements of the eyes, nystagmus, paresis of the limbs and of the trunk muscles, loss of tone in the muscles, ataxia of movements, tremor, staggering gait, alteration of the deep reflexes, with normal superficial reflexes, and peculiar fits." In both extra-cerebellar and intra-cerebellar growths the headache is severe, vomiting is nearly always present, and optic neuritis is, perhaps, more early in its appearance in intra- than in extra-medullary tumors. Of the localizing symptoms, vertigo or giddiness is a frequent symptom in both forms of tumor.

"Taking, first, the extra-cerebellar tumor, we find that the majority of them grow from the flocculus or the auditory nerve in the anterior part of the posterior fossa. They are wedged in the angle between the cerebellum and the pons close up to the petrous part of the temporal bone and compress these structures without actually involving them. In these tumors we find that compression of the cranial nerves, and especially of the auditory and facial nerves, is a very common symptom, and as a result we get loss of hearing to sounds heard through the air, as well as those conducted by bone, also tinnitus, buzzing noises; facial paralysis involving all parts of the face; occasionally the fifth nerve, the trigeminus, is pressed on, and especially the sensory part, causing numbness, tingling, or loss of sensation of the face on the same side as the tumor; also in many cases paralysis of the sixth nerve with weakness

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(1) Lancet, Mar. 16, 1907.

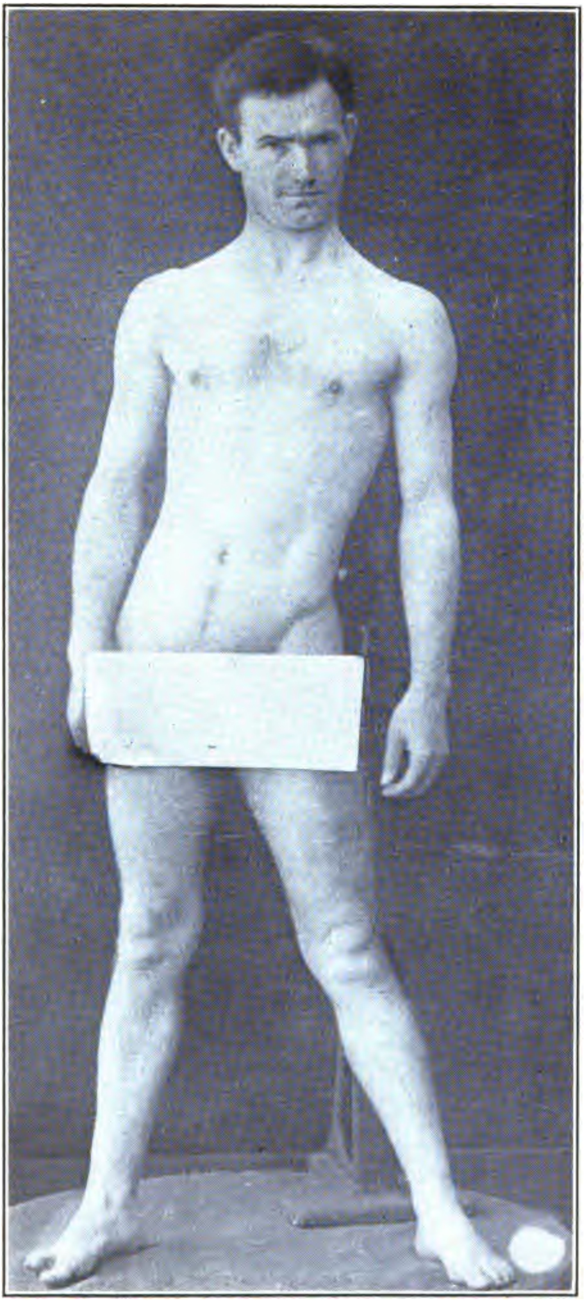

PLATE II. Attitude in cerebellar disease (sclerosis). (Page 126.)

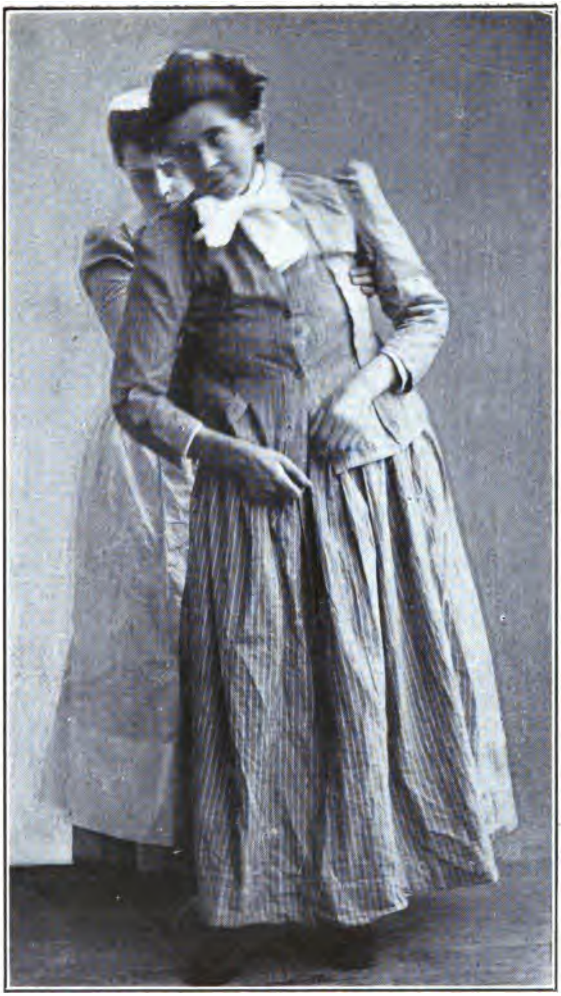

PLATE III.

Attitude in cerebellar disease (Fraenkel), tumor of pontine angle. (Page 126.)

of the external rectus; rarely the ninth, tenth, and twelfth nerves. This compression of the cranial nerves does not occur in intra-cerebellar tumors. Motor symptoms occur on the same side as the lesion. This shows itself in the conjugate movements of the eyes, and nystagmus. This occurs in both forms of tumor, perhaps more in extra-cerebellar. In the limbs on the same side as the lesion in intra-cerebellar lesions there is diminished power, and in a few cases the muscles of the spinal column, especially the erector spinæ, of the same side are weak. Associated with the hemiparesis there is loss of tone, so that the muscles of the limbs on the same side of the lesion are flaccid and flail-like. Associated with incoördination is the condition pointed out by Babinski in the paretic limbs of the same side, viz., that the patient cannot rapidly pronate and supinate the forearm owing to a defective coördination of the muscles.

"The condition of the reflexes is one of much importance, and especially the knee-jerk. In extra-cerebellar tumors the knee-jerk is not altered, while in intra-cerebellar tumors of one side the knee-jerk on that side is, if anything, diminished. The superficial reflexes are not altered in intra-cerebellar tumors, and I have never seen the extensor plantar reflex in pure cerebellar cases.

"It must, however, be remembered that, in extreme pressure from an extra-cerebellar tumor on the pyramidal tract in the pons, paresis may occur in the limbs on the opposite side, due to decussation of the pyramidal tract below, but when this is the case there will be some rigidity with increase of the knee-jerk, diminution of the superficial reflexes, and the extensor plantar reflex in the opposite limb.

"The diagnosis of cerebellar tumors from cerebral is made by a comparison of the symptoms given above with those found in tumors of the cerebrum. The absence of mental symptoms, of sphincter troubles (except at the end of the disease), and of epileptiform attacks, the presence of a reeling gait associated with deviation, say, to the left, with weakness of movements of the eyes to the left and paresis of the left limbs without alteration of the reflexes, would be in favor of an intra-cerebellar tumor of the left lateral lobe; while deafness and facial paralysis of the left side with conjugate weakness of the eyes and paresis of the limbs of the left side, and with weakness and rigidity of the right limbs, together with increased knee-jerk and extensor plantar reflex, would be due to an extra-cerebellar tumor of the left side."

B. Sachs[1], in discussing the symptoms of cerebellar tumor, says that "they present, first of all, the symptoms common to all intracranial neoplasms, viz., headache, nausea, and vomiting, double optic neuritis. As for the last, it is to be noted that the optic neuritis appears almost invariably at an unusually early stage of the disease, that it leads with unusual rapidity, in many instances, to complete amaurosis, and that this physical sign is wanting less often in cerebellar than in other intracranial neoplasms. Other things being equal, rapidly advancing optic neuritis may serve as a symptom pointing to cerebellar tumor, for I have found this rapidly progressive form of double optic neuritis more regularly in cerebellar neoplasms than in tumors of the frontal lobe, and also more frequently than in cases of tumor in the temporosphenoidal region, or in the motor areas.

"The early development of unilateral or bilateral rectus externus palsy is to be given due weight. I have been accustomed to regard it as almost pathognomonic of cerebellar lesions if it be associated with the general symptoms pointing to increased intracranial pressure. No doubt the peculiar position of the abducens nerve, the fact that it may be compressed very readily between the brain-substance and the base of the skull, will account for the frequent occurrence of this special form of palsy. An irregular lateral nystagmus is often observed in these cases on extreme outward movement of the eyes.

"Authors are not yet agreed as to the importance to be attached to the side to which the patient sways in walking or standing. The evidence from my own cases would seem to be largely in favor of the patient swaying to the side in which the tumor is situated, but I would not allow any evidence of this sort to persuade me to determine the site of operation if other signs point to the opposite side of the brain." * * * "Changes in the electrical reaction of the facial nerve often setting in before a marked facial

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(1) Med. Record, Dec. 22, 1906.

palsy has been developed, have helped to corroborate a suspicion of cerebellar disease, and have at the same time pointed to the exact site" in the cerebello-pontine angle. Early involvement of the auditory nerve is of similar value in diagnosis and localization. "If the progress of the disease is an unusually slow one, and if facial, auditory, and possibly trigeminal symptoms are present; if, moreover, in such cases the symptoms are unilateral or bilateral, the diagnosis of a neurofibromatosis may be entertained.

Tumor of the Cerebello-pontine Angle. Oppenheim and M. Borchardt[1] report two cases of tumor at the cerebello-pontine angle with operation. In the first case the leading symptoms were occipital headache, vertigo, vomiting, nerve-deafness in the right ear, cerebellar ataxia, double choked disk, nystagmus, limitation of vision to the right, absence of the right corneal reflex with slight transitory disturbances of sensation in the right half of the face, paresis of the right facial nerve with quantitative reduction of its excitability, paresis and reduction of the palatal reflex, slight ataxia of locomotion, and diadokokinesia [See PRACTICAL MEDICINE SERIES, Vol. X, 1903, p. 162—ED.] in the right arm and to a lesser extent in the right leg. The tumor was located in the right cerebello-pontine angle, and was removed by Borchardt. A second case of greater severity, completely blind before operated upon, was likewise restored to health, but of course his sight remained absent. The technic of operation is carefully described by Borchardt, and the description is accompanied by one figure, illustrating the very extensive osteoplastic flap, consisting of practically the whole of the lower part of the table of the occipital bone.

Two cases of tumor at the cerebello-pontine angle, with the postmortem findings, are reported by Theodore Diller.[2] The first case, a young man, aged 29, showed slight nystagmus, an internal squint in the left eye, deviation of the tongue to the left, slight defect in enunciation, dribbling of saliva when eating or speaking, and absence of the sense of taste entirely and of touch almost wholly over the

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(1) Berlin klin. Woch., July 15, 1907.
(2) Jour. Am. Med. Assoc., July 27, 1907.

left half of the tongue. On the right side of the tongue touch and taste were present but impaired.

The patient masticated on the right side exclusively. He was deaf in his left ear. The knee-jerks were much increased, and equal. The Babinski toe-sign was present on the right side. There was no ankle-clonus. Motion was somewhat impaired on the right side. He could not stand alone, and when assisted in walking, lunged backward and forward and sidewise in an uncontrollable fashion. Subsequently he began to vomit frequently, vision rapidly failed, a slight paralysis of the left face was apparent. An attack of suspended breathing occurred May 19, 1905, but artificial respiration restored him to consciousness. Eight or ten similar attacks followed, in the last of which he died. The tumor, about the size of a hickory nut, was found postmortem growing from the anterior margin of the left cerebellar hemisphere close to and pressing on the pons, which was deeply indented to accommodate it. The upper portion of the oblongata was also pushed over and flattened by pressure from the tumor, but to a lesser degree than the cerebellum.

"In the presence of headache, vomiting, vertigo, and staggering a cerebellar tumor was indicated; and it was supposed to be at the left cerebello-pontine angle because of the involvement of the cranial nerves on the left side from the fifth to the twelfth inclusive. The marked involvement of the left fifth and eighth nerves, together with the cerebellar symptoms, especially indicated this diagnosis.

"The situation of the tumor with its direct and indirect pressure readily explains the involvement of the last cranial nerves or their nuclei on the left side. But why the function of the eighth nerve should be totally destroyed and the seventh be only slightly affected, since both nerves arise closely together at the lower border of the pons, is an interesting question which I am unable to answer."

The second case, a woman, aged 42, suffered from headaches, very severe at times, and with severe vomiting. The headaches were at the top of the head and in the upper frontal region, and not localized on one side especially. Spells of unconsciousness of from six to eight minutes' duration were frequent, often, but not always, accompanied by jerking, usually in the left arm and left leg, but in the right arm and leg at times. These attacks came on anywhere at any time. Defect of vision followed, and the disks were swollen, showing also many hemorrhages. She also became deaf in the left ear. The location of the tumor was undetermined, but was found postmortem to be the size of a hulled walnut, located at the left cerebello-pontine angle.

Treatment of Cerebellar Tumors. B. Sachs[1], of New York, in some notes on the diagnosis and result of surgical treatment of cerebellar tumors, has contributed a very able article to the literature of this subject, and he illustrates his remarks by carefully selected clinical cases. The question of the surgical removal of cerebellar tumors is still an open one. "The difficulties of diagnosis are somewhat greater than those commonly met with in the case of other intracranial neoplasms. Not infrequently the diagnosis is made by exclusion, and strictly localizing symptoms are less certain than they are, for instance, in the cases of tumors, affecting the motor or occipital areas." Sachs bases his notes upon twenty-two cases, sixteen seen in hospital, and six in private practice.

Operation was urged in only seven of these, and in two of these the tumor was not found, though it was proved to be present by autopsy. These two cases are reported in full. In both of these cases the localization was nearly accurate; but even if the surgeon had gone deeply into the cerebellar tissue, the tumor could scarcely have been reached. The danger from hemorrhage would have been infinitely greater, and as it was, the second patient of these two died from respiratory paralysis, probably due to secondary hemorrhage.

Of the seven cases operated on, two are already accounted for. A third patient died a few days after the operation. In the four remaining cases a simple trephining operation was done over one or both lobes of the cerebellum, and in these patients the operation was followed by marked improvement, or at least temporary relief.

In sixteen patients operation was not advised, "either because there seemed to be no possibility of finding the tumor, or because the tumor was supposed to be on the

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(1) Med. Record, Dec. 22, 1906.

ventral surface, or because the symptoms were too advanced, or were not so pressing that operative interference was imperatively demanded."

Though the routine administration of iodids and mercurials seems unscientific, yet Sachs confesses that in some instances much was gained by such treatment, three of his cases, not included in the twenty-two above, presenting symptoms pointing to cerebellar neoplasm having recovered. In but one of the three was there a distinct specific history. On the other hand, in cases in which the headaches are intense, the gait growing progressively worse, the optic neuritis steadily increasing, trephining should be at once advised, for the neoplasm may possibly be capable of being removed, and blindness may be prevented. The author's concluding paragraph follows:

"While I firmly believe that the cure of cerebellar tumors is difficult to attain, except in a very small proportion of the cases, I believe that when the diagnosis of cerebellar tumor is made, and when there is a fair degree of certainty as to the special part of the cerebellum which is involved, a large trephine opening should be made over the suspected area; and, even if at the first or at the second stage of the operation the neoplasm is not found, much good will be done by the relief of pressure, the headaches will disappear, or will at least be diminished in severity, and blindness—the greatest of all scourges in these cases—may thereby be prevented or considerably deferred."

MEDULLA OBLONGATA.

Devic and Nordmann(1) relate a peculiar case. A patient of 48 years of age, a confirmed drunkard for twenty years, ultimately died of delirium tremens, cirrhosis of the liver and tuberculous pleurisy. Aside from the nervous symptoms of delirium tremens, the only signs pointing to impairment of the nervous system was a history of painful paresis of the lower extremities. Since this was associated with lost knee-jerks and pain on pressure of the muscles of the calf, the diagnosis of an alcoholic peripheral neuritis was suggested.

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(1) Lyon Med., July 22, 1906.

At the postmortem examination a rusty pin or needle (it was impossible to say which) was found, one end free, and the other sticking into the medulla about two-fifths of an inch. It had entered a little to the left of the median line, about 2 to 3 millimeters above the back of the calamus scriptorius. It passed from left to right, downward and forward. The coat of rust proved that it had been in the place in which it was discoveerd for some time, as did the state of the nerve-tissue immediately adjoining it, this being slightly softened and of the same ochre color as the surface of the needle itself. Yet notwithstanding this rusty bit of needle or pin, the patient had no discoverable symptoms from it in life.

DISEASES OF THE SPINAL CORD.

Theory of Conduction of Sensory Impulses. J. Babinski[1], in presenting a case of unilateral lesion of the medulla, showing thermo-asymmetry, vaso-asymmetry, and hemianesthesia of the syringomyelic type, advances what he called a new hypothesis as to conduction of the different forms of sensation, but which was really first advanced by Long as early as 1898. Babinski calls attention to the fact that in syringomyelia the dissociation consists in the preservation of touch and the loss of perception of pain and temperature. He observes that the reverse of these losses, loss of tactile sense and preservation of the senses of pain and temperature have never been observed. Why not? If there be differentiation in the cord, why this curious fact? If, on the other hand, the cord indifferently conducts sensory impulses upward through the gray matter, slight injuries might give the loss of pain and temperature sensations with the preservation of the tactile sensory impulse conduction.

At the end of a most lengthy and painstaking article on the different impulses within the spinal cord by Head and Thompson[2] is an important review of the whole matter. Bearing on Babinski's remarks as to conduction of nerve impulses is the following: "From the above, and similar,

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(1) Rev. Neurol., Dec. 15, 1907, p. 1177.
(2) Brain, Part IV, 1906, issued Mar., 1907.

observations it follows that in the dog and lower animals no one form of apparent impulse is restricted to any one tract, nor even to one-half of the spinal cord. For each, more than one path seems open, and can with time be rendered available. The sense of touch seems most localized and passes in the homolateral posterior column and heterolateral anterior column, whilst, for instance, pain-impulses pass chiefly in the homolateral, though also in the crossed lateral column, and possibly also in the anterior columns; it appears that the different factors underlying muscular sensibility can pass in addition along the posterior columns."

TRAUMA OF THE SPINAL CORD.

Alexis Thomson[1] discusses the differentiation of partial from total transverse lesions of the spinal cord, and reports two illustrative cases. In total transverse lesions every function of the cord below the site of injury is immediately and permanently arrested. The only sign of self-action remaining in the isolated part of the cord is the occasional presence of the skin reflexes in much reduced degree. Without exception there is complete paralysis of the bladder and rectum. While Kocher would lead one to infer that he associates priapism with total lesions, Thomson inclines to the view that its presence is quite compatible with partial ones. The return of the deep reflexes at any subsequent period signifies that the lesion has not remained total, and that conductivity through the region of the lesion has again become possible, either from recovery of elements of which the function has been in abeyance for some time and has been resumed, or the taking over of their functions by the undamaged fibers.

"When there is recovery from an apparent total transverse lesion, the first sign of such recovery is the return of the knee-jerks and a gradual change in the limbs from the flaccid to the spastic condition. Sensibility returns in the order—touch, pain, temperature,—and the parts supplied by the lowest sacral segments usually become sentient before those supplied from a higher level. Sometimes the first sign of such return is the patient becoming conscious of the act of micturition or of catheterization or of disten-

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(1) Edinburgh Med. Jour., July, 1907.

sion of the bladder during the process of irrigation (Collier). Voluntary power returns earlier in the flexors than the extensors, and flexion of the toes is almost invariably the earliest voluntary movement possible.

"When one is called to a case of injury to the spine with complete abrogation of all the functions of the cord below the seat of injury, it is impossible to determine whether the abrogation is due to destruction of all the nerve elements—a total transverse lesion—or whether there is only partial destruction of the nerve elements with temporary abrogation of the functions. One has not long to wait before the question is cleared up; if the lesion is a partial one, the deep reflexes return within a few days of the injury. On the other hand, cases are reported in which the return of the knee-jerks has been delayed for a month or six weeks."

The first case which Thomson reports was that of a coachman showing a lesion limited to the first dorsal segment of the cord, the segment appearing to be completely destroyed, as indicated by the symptoms presented, yet the lesion in reality was due largely to extravasation of blood into the substance of the cord, as shown by his partial recovery. The onset of the paralysis was abrupt and absolutely complete at the time of accident, and because of this hemorrhage and pressure upon the cord were excluded in the differential diagnosis. The completeness of the paralysis and its exact correspondence to a total transverse lesion of the first dorsal segment led to the diagnosis of a total destruction of the cord at this level and of a bad prognosis. Yet in time the man walked again.

"The practical lessons to be drawn from the case are, firstly, that, given the abrogation of all the functions of the cord, one must not allow oneself to be committed to the diagnosis of a total lesion until a period of some five or six weeks have elapsed since the accident; and, secondly, to decline operative interference unless there is reliable evidence that there is pressure upon a cord which is not irreparably damaged, and that the cause of the pressure can be removed by operation. To be of service, the operation must be performed within seven days of the occurrence of the lesion."

ARTERIOSCLEROSIS.

Joseph Collins[1], in a general article upon arteriosclerosis of the central nervous system, says that arteriosclerosis of the spinal cord may give rise to many and very different symptoms, depending upon the segment of the cord that is involved, and upon the components of the cord that are encroached upon in transverse section. He has, in a series of 800 cases of arteriosclerosis affecting the nervous system, seen not less than six autopsies in which the cord by careful microscopic examination has been found affected. The diagnosis of transverse or chronic myelitis is confidently made, and is found at postmortem to be wrong. The onset may be very abrupt, and in one case the diagnosis of Landry's paralysis was made.

TABES DORSALIS.

Etiology. S. Filipkievitz[2] discusses tabes dorsalis and syphilis, basing his remarks on 328 cases. Of these 304 had syphilis beyond peradventure of a doubt. Of these he himself had treated 35 for the original syphilis; 266 others were treated for syphilis by his colleagues, and 3 showed symptoms of active syphilitic disease with their tabes. In 300 cases syphilis had been acquired; in one case it was inherited. The percentage of tabetics with syphilis is, therefore, 92.6 per cent. In 258 of the 300 cases the original treatment was badly carried out. The author concludes that syphilis is the chief factor in the causation of tabes, but that it is not the only factor; and that insufficient treatment of the original condition is more likely to be followed by tabes than thorough treatment; that besides syphilis, there must be some other unknown factor, which, united with syphilis, is capable of producing the disease.

W. Spielmeyer[3] has produced an *experimental tabes dorsalis* in dogs by means of trypanosomes, in every way identical with that produced in man by syphilis.

Symptomatology. As to symptoms, S. Filipkievitz[4] presents the following incidence:

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(1) Southern Med. and Surg., July, August and September, 1906.
(2) Pester Med.-Chir. Presse, May 26, 1907.
(3) Münch. Med. Woch., Nov. 27. 1906.
(4) Pester Med.-Chir. Presse, May 26, 1907.

	Per cent.
Lancinating pains	86.0
Girdle sensation	29.0
Sensory disturbances	78.0
Gastric crises	7.0
Laryngeal crises	0.3
Intestinal crises	3.6
Bladder disturbances	52.0
Sexual disturbances (males)	30.0
Ataxia	33.0
Romberg's Sign	66.0
Transitory diplopia	5.0
Argyll-Robertson pupil	17.0
Unilateral dilatation of pupil	41.0
Inequality of pupils	18.0

	Per cent.
Ptosis	7.6
Trigeminal anesthesia	4.8
Paresis of the facial nerve	0.9
Paresis of peroneal nerve	1.0
Arthropathies	8.8
Necrosis of inferior maxilla	0.3
Absent knee-jerks	75.0
Diminished knee-jerks	10.0
Unequal knee-jerks	7.8
Loss of one knee-jerk	5.1
Increase of knee-jerks	1.5
Optic atrophy	3.6
Oculomotor paralysis	6.0

Paul Camus and A. Sézary[1] presented at the December meeting of the Neurological Society of Paris a case of cervical tabes showing *unilateral ataxia,* limited to a single limb. The lesion seems to have involved certain well defined rootzones, as indicated by the ataxia found in the left arm and the astereognosis of the left hand.

Tabetic Joints. L. F. Barker,[2] discussing joint affections in nervous disease, refers at some length to tabetic joints. In Charcot's original description the main clinical features were the sudden appearance of a firm, painless swelling of a joint, extending to the surrounding soft parts, occurring often independently of traumatism, followed by rapid changes in the joint surfaces leading to dislocations and subluxations and to false positions of the bones in a very short time. Pain, while usually absent, is sometimes present. In Henderson's analysis it occurred in only 20 out of 333 cases. The joint is rarely red, though in some instances slight redness appears and lasts for a few days.

"The sudden firm swelling is very characteristic. It does not pit on pressure and is quite different from an ordinary edema. It extends with great rapidity, often reaching its height in a few hours, and involves structures beyond the capsule and bursæ of the joint. Occasionally crepitation can be made out very similar to that met in ordinary arthritis deformans.

"Tabetic arthropathies may occur at any stage of the disease. They may even be among the premonitory symptoms, though this is unusual. The majority of the cases appear to have been observed first in the ataxic period. In

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(1) Rev. Neurol., Dec. 30, 1907.
(2) Jour. Am. Med. Assoc., Feb. 2, 1907.

a total of 246 patients analyzed by Henderson, the first arthropathy occurred in the pre-ataxic stage in 54 cases, in the transitional in 36 cases, in the ataxic in 156. An arthropathy which might have remained benign had the joint been kept at rest is often converted into a malignant arthropathy by neglect and overuse. The painless character of the affection tends to lead patients to use the joint, unless they are strictly warned against this by the medical attendant. Almost any joint in the body may be affected, though the small joints appear to be less often involved than the large. Even the maxillary joint may become affected."

Pregnancy in Tabes. P. Zacharias[1] reports a case of *pregnancy* in tabes dorsalis in a patient who first showed symptoms of tabes nine years ago. Six years ago she bore a child without much pain. She lately became pregnant again, despite signs of well-marked tabes, such as diplopia, ataxia, lightning pains, sensory disturbances, lost knee-jerks, and Argyll-Robertson pupils. During the last month of pregnancy she fell, breaking the neck of the right femur. Delivery was painless, though the uterine contractions were powerful. The authentic cases of delivery of normal children in tabetics are very few. There is the case of Litschkus and that of Mirabeau, and cases of Bernhardt, P. Müller, Cohn, and Thies, besides this case reported by Zacharias. Most of these labors were rapid and painless. [The author omits the three cases of Jean Heitz summarized in the PRACTICAL MEDICAL SERIES, 1903, Vol. X, p. 198.—ED.]

Walker G. Bowers[2] reports a case of *tabes dorsalis in a negress.* Bowers has collected 21 reported cases of tabes in the colored race, 15 being males, and 6 females. Clinically and by postmortem examination the diagnosis of tabes dorsalis was incontrovertible.

Treatment. For those wishing special information as to the treatment of tabes, attention may be called to the hydrotherapeutic treatment of that disease as set forth by A. Laqueur,[3] based upon 135 cases treated at the hydro-

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(1) Münch. med. Woch., Feb. 12, 1907.
(2) New York Med. Jour., Oct. 27, 1906.
(3) Berlin klin. Woch., Mar. 4 and 11, 1907.

therapeutic-medico-mechanic institute at Berlin, connected with the University of Berlin; and also to the physical treatment of tabes as set forth by E. Tobias[1] and E. Kindler. The latter authors consider fully the diet, massage, gymnastics, the compensatory exercises of Frenkel, hydrotherapy, and electrotherapy, their article being a thorough review of the whole subject, without any new suggestions, however.

ACUTE ASCENDING MYELITIS.

E. Farquhar Buzzard,[2] in the Goulstonian Lectures, discusses acute ascending myelitis in a general article on acute infective or toxic conditions in the nervous system. He prefers the term 'ascending' to 'disseminated,' since the former appellation graphically suggests the prominent characteristic of the disease. His remarks are based upon the case of a man, 44 years of age, who on Aug. 3, 4 and 5, 1903, noticed dull pain in the chest, especially between the shoulders. On the following day he felt weak in the legs, and by August 7 could not empty his bladder. On the ninth he felt no pain when pins were jabbed into his legs, and on the twelfth suffered from pains shooting down his arms, followed by paresis of those limbs in 24 hours. On the sixteenth he died from respiratory failure, and 12 hours after death Buzzard made the postmortem examination. The arachnopia was intensely congested, but there was no exudate. "The spinal cord was softer than usual throughout its length, but from the tenth dorsal segment to the lower cervical region almost diffluent in consistence. On transverse section in this part it was impossible to distinguish white from gray matter and dark brown patches suggestive of extensive hemorrhages were seen scattered about. Beyond general hyperemia the brain presented no abnormal features. Part of the cerebrospinal fluid taken during life was centrifugalized and examined. It contained few cells but some organisms, best described as diplobacilli—very short rods with rounded ends placed end to end, generally in pairs but sometimes forming irregular chains. Cultures from the cerebrospinal fluid were sterile. Cultures from the venous blood extracted during

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(1) Brain, Part 117, June, 1907.
(2) Lancet, Mar. 16, 23 and 30, 1907.

life produced in broth only a slight turbidity and smears showed a diplobacillus resembling the one already described. All efforts to subcultivate the organism failed. All culture tubes inoculated postmortem proved sterile except the broth tube from the spinal-cord substance which again grew a scanty and short-lived organism resembling the one obtained from the venous blood during life. The organism referred to retained Gram's stain badly."

We may therefore assume, continues Buzzard, that acute ascending myelitis is an infective disease probably due to the presence of various bacteria in the tissue of the cord. As to the pathogenesis, there is strong evidence that the disease is the result of bacterial infection of the *lymphatic system* of the spinal cord. "In acute ascending myelitis we have a condition closely resembling the morbid process produced experimentally by Homen when he injected organisms into the sciatic nerve or into the substance of the spinal cord. The characteristics common to both the human disease and the experimental results are the scattered irregularity of the lesions, the preference for the white matter, and especially for the posterior columns, the spread of the process in an upward direction along the central cord and meninges, in addition to the essential features of the inflammatory processes.

"The conclusions are as follows:

"1. A group of cases, which have the same clinical picture, but which may arise either spontaneously or as a secondary complication in other infective conditions, deserves the name acute ascending myelitis.

"2. These cases are characterized from the clinical point of view by an acute ascending paralysis associated with marked loss of cutaneous and deep sensibility of all kinds and with loss of sphincter control, and anatomically by an acute inflammation of the spinal cord, affecting both the white and gray matter, beginning generally in the lower dorsal or lumbar regions and spreading upwards to the medulla.

"3. Both on experimental and clinico-pathologic grounds there is evidence to support the view that the inflammation is due to an infection of the lymphatic system of the spinal cord; in other words, the disease is a spinal lymphangitis.

"4. The condition is not a specific one and may be due to the action of various micro-organisms. There is no particular age incidence nor is there any instance of an epidemic tendency."

ACUTE POLIOMYELITIS.

E. Farquhar Buzzard[1], in the Goulstonian Lectures, discusses acute poliomyelitis. He advisedly omits the word "anterior" usually employed, since it describes a limitation at times incorrect. The word "infantile" should also be omitted because the disease is met with in any of the first four decades of life, and cases in adults are being observed with increased frequency. Buzzard's basis for his study was a man dying within four days of the first symptom of illness and within 36 hours of the onset of paralysis. The following summary is made by the author from his own findings, as well as those of others, in cases examined within a few hours or days of the onset of the disease: "1. The most widespread and constant features are, on the one hand, the congestion of blood-vessels and proliferation of the cellular elements of their adventitial sheaths; on the other hand, the absence of all evidence of ante-mortem thrombosis or of changes in the intimal coat. 2. An intense proliferation or exudation of various cells, concerning the origin of which there is a difference of opinion, limited largely to the neighborhood of the rich capillary network of the gray substance of the cord. 3. Round-cell infiltration of the meninges more marked in the lower parts of the cord and on its anterior aspect, but extending in some cases to the intracranial meningeal cisterns. 4. The morbid anatomy of the epidemic and sporadic cases, whether in children or adults, has always proved to be essentially the same in the acute stage."

The evidence that acute poliomyelitis is of an infective nature is chiefly clinical. The *symptoms* are, generally speaking, the onset of paralysis, preceded by fever, vomiting, convulsions, general malaise and pains in the head, neck, back, and limbs. In the more fulminating type of case rigidity of the muscles of the neck and back has been

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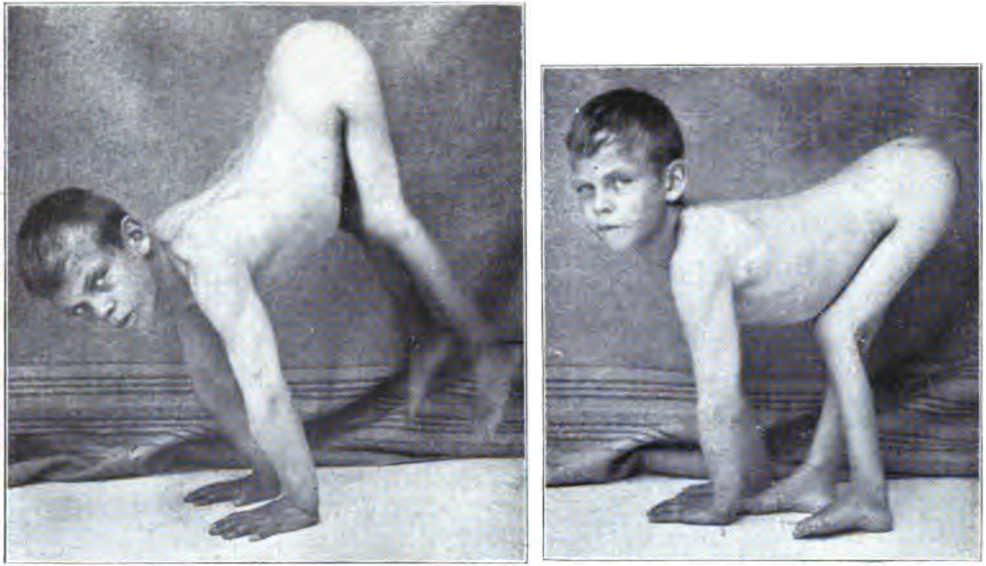
(1) **Lancet, Mar. 16, 23 and 30, 1907; also Brain, June, 1907, No. 117.**

noted. In the infant vomiting and convulsions are common; in the adult they are rare, though cases are recorded. The older cases give us our information as to pain, and some observant mothers have noticed that children cannot bear to have their limbs handled in the earliest stages. The pain may be spontaneous or produced by movement. The paralysis of onset is always more extensive than the residual paralysis on recovery. Another clinical factor of importance is the occasional presence of the Babinski toe-sign, which Buzzard has obtained in several cases. It is, of course, in only a small number of cases that the toe-muscles are left intact, and, moreover, it is difficult to distinguish between a pathologic extension and an infantile wiggle; hence the toe-sign test is rarely applicable. When present it affords evidence that there exists definite involvement of the pyramidal tracts.

"Another clinical point is worthy of note. In epidemics and in family cases, other members of the household have been taken ill at the same time without developing paralytic phenomena. There is evidence, therefore, of the existence of what may be termed abortive cases. On the other hand, there is a great probability that many cases of acute generalized poliomyelitis occur in young infants and rapidly prove fatal without being recognized."

As to pathogenesis, Buzzard affirms that "we are in position to say that the process is an inflammatory one and probably due to the influence of some micro-organism or its toxin.

"The following conclusions appear justifiable concerning the disease: 1. It is an acute specific fever occurring sporadically and epidemically, differing from many others but resembling some—variola, for instance—in the facts that its lesions may be discrete or confluent and that its results may be permanent. 2. Its essential lesion is an inflammation of the interstitial tissue of the central nervous system due to the presence of micro-organisms or their toxins probably in the blood (but possibly in the lymph) circulating within that system. 3. The fact that the gray matter suffers more than the white matter is explained by the greater vascularity of the former and the resistant character of the component parts of the latter. 4. The same morbid process underlies some cases of



A. **Anterior poliomyelitis.** B. **Anterior poliomyelitis.**

PLATE IV.

Anterior poliomyelitis. **Involvement** of both lower extremities. Extreme flexion deformity at hips. Quadrupedal attitude. Upper extremities developed to high degree of muscular strength. Simian appearance. Double knock knee. Treatment.—Arthrodeses of ankle joints. Transference of sartorius to quadriceps tendon on left side. Exsection of contracted ilio-tibial bands. Double osteotomy for knock knee. Apparatus and crutches. (Page 146.)

encephalitis both of the brain stem and the cerebrum, among which may probably be included many examples of infantile hemiplegia. 5. The bacteriology is at the present time practically an unknown quantity."

Stanley Barnes and James Miller[1] report a case of acute poliomyelitis in a boy, 9 years of age, together with the postmortem and bacteriological findings. In seven of the tubes after 24 hours there was a slight amount of growth which proved later to be *Staphylococcus pyogenes citreus,* and in two of these tubes there were in addition single colonies of *Staphylococcus albus.* The authors regard their case as one of acute generalized poliomyelitis of the same type as that usually called infantile paralysis. The anatomical findings indicate that the changes in the gray matter of the cord are inflammatory and not thrombotic. The disease *may* have a bacterial origin. In this disease parts of the gray matter of the cord other than that of the anterior horns may be involved.

In the same issue J. J. Perkins and L. S. Dudgeon[2] report acute poliomyelitis in an adult, with marked bulbar and ocular symptoms, and they also publish the postmortem findings in their case. These findings agree with those of Buzzard in the Goulstonian Lectures, and bear out his conclusions. The diffuseness of the lesions, not confined to the large cells of the gray matter, and in the white matter causing peripheral fatty degeneration and affecting severely the neuroglia, was well shown. The whole nervous system was involved, including the brain. Indeed, in this case, the lumbar symptoms were the most marked feature of the case clinically. Bacterial examination was negative, but formalin had been injected into the spinal canal.

A case of *relapsing acute poliomyelitis* by Reginald Miller was reported in the same issue of *Brain* (Part 117). The points of special interest in this last case are, first, the number and nature of the relapses that occurred; secondly, the extremely widespread distribution of the foci of disease found in the central nervous system at the postmortem examination. The case occurred in a little boy 3½ years old. Pathologically it supports the hypothesis that the disease is primarily inflammatory and not vascular.

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(1) Brain, Part 117, June, 1907.
(2) Brain, Part 117, June, 1907, page 110.

L. Pierce Clark and C. E. Atwood[1] report two cases of acute anterior poliomyelitis *with exaggerated knee-jerk,* one in a boy of 13 years, and the other in a girl of 3. In the boy the left knee-jerk was exaggerated from the very onset of the disease.

Treatment. Nathaniel Allison[2] discusses the relative value of the surgical procedures employed for the relief of paralysis following acute anterior poliomyelitis. The surgical procedures in vogue are (1) tendon-transplantation, with its subdivisions of muscle-insertion, muscle-transference, muscle-shortening, tendon-shortening, tendon-lengthening, tendon-transplantation, the periosteal insertion of tendons, and the added method of lengthening tendons by silk sutures; (2) arthrodesis, alone or with tendon-transplantation, having for its object the securing of such stability in a flail-like joint as will enable the patient to progress with little or no apparatus; and (3) nerve-transplantation, by which healthy nerves are transplanted into degenerated nerve fibers going to atrophied and paretic muscles. The particular procedure to be selected depends entirely on the case. In the extreme cases, with the flail-like joints, without muscular power, the best that can be accomplished is to make of the paralyzed limb a limb similar to an artificial limb, that is, one which will bear weight through stiffening by an operative procedure. Children so affected assume quadruped positions, as shown in Plate IV, A and B, and Plate V, B. If the lower extremities are particularly involved, the upper ones take on the function of weight-bearing and progression. "For these cases it is necessary to employ apparatus such as will stiffen the lower extremities, that they may be used as props, so that the individual may assume an upright position, supported by crutches. The assumption of the upright position is of the greatest importance to the mental attitude of these cases. (See Plate V, A; and Plate VI.)

"Next to this extreme class of cases are those less severely affected, where, for instance, most of the muscles of the leg are involved, the muscles of the thigh remaining powerful enough to be of use in walking. This class of cases usually does well with braces. Operative procedure, however, is justifiable, in that by means of it we may discard the wearing of braces. Such cases are much benefited by the employment of arthrodeses, or the similar operation employed at the ankle, of exsecting the astragalus. By means of these operations the limb may be stiffened so that it does not give way under superincumbent weight, and the muscles that remain may place it in positions favorable to progression."

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(1) New York Med. Jour., July 20, 1907.
(2) Interstate Med. Jour., September, 1906.

The third division of cases is made up of those in which certain muscles in an extremity are lost, or weakened, but in which the remaining muscles are of normal power. "The problem in this class of cases is to utilize the muscles either by transferring their tendons, or grafting their muscle-bellies into the tendons or muscle substance of the weakened or lost muscles. The forms of paralysis which have been most benefited by tendon-transplantations are those which involve the foot. The transplantations of a portion of the tendo Achillis to the extensor part of the foot for the relief of equinus have also shown distinct improvement. The transplantation of the peronei to the inner side of the foot for the correction of valgus deformity have not been attended by good results. At the knee, the use of the sartorius as transplanted into the quadriceps extensor tendon has frequently resulted in power of extension of the leg on the thigh. The transplantation of the trapezius into a paralyzed deltoid has shown uniformly good results.

"*Conclusions.*—(1) In extreme forms of infantile paralysis the wearing of apparatus and the use of crutches is necessary, that the upright position may be maintained. (2) In extreme forms of infantile paralysis, the wearing of heavy appliances may be materially lessened by the employment of operations designed to fix the joints. (3) In cases where there is good muscular power remaining in strong muscles, these muscles may be used to good advantage by transference of their tendons. Care should be taken that the insertion of these tendons is strong and permanent, and that the muscles pull to the best mechanical advantage. (4) In certain cases, tendon-transplantation may be supplemented by arthrodeses to good advantage. (5) In slight forms of paralyses, the grafting of nerves is a surgical procedure of recognized merit."

LANDRY'S PARALYSIS.

E. Farquhar Buzzard[1], in the Goulstonian Lectures, discusses Landry's paralysis. His first question is whether there is such a distinct morbid entity. Probably no other title has been so abused as this, for under it have been described every form of paralysis of rapid onset, beginning in the lower extremities and extending upward. Nearly as many fatal cases of acute poliomyelitis in adults have been published under the name of Landry's paralysis as under their proper title. Again, on the other hand, strenuous efforts have been made by some to force all cases of Landry's paralysis into the category of peripheral neuritis. "To my mind it does not matter, nor shall we ever know, what was the actual disease from which the cases described by Landry suffered. It is sufficient that he called our attention to a class of case in which the symptoms were those of a rapidly spreading motor paralysis without atrophy and without sensory or sphincter disturbances, and in which he was unable by the methods at his disposal to detect anatomical changes."

Buzzard reports briefly three cases which he examined clinically and anatomically in which the symptom-complex described by Landry was present. In all three cases the pathologic findings were identical, and they may be summarized as follows:

"1. No evidence of disease was present in the nervous system to the naked eye, except vascular engorgement. It is particularly to be noted that the cord was firm in consistence throughout in all cases.

"2. The only cell change was that seen in the spinal cord by the Nissl method, and in no instance was it extensive.

"3. By the Marchi method the nerve fibers in the cord and in the peripheral nerves showed the fine scattered droplets of fat. This form of myelin degeneration differs from true Wallerian toxic degeneration in the fact that the droplets are smaller and discontinuous, generally single or only two or three together, and not necessarily associated

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(1) Lancet, Mar. 30, 1907.

with any loss of function on the part of the axis cylinders. Having found a similar appearance in all sorts of toxic conditions without paralysis, I am not inclined to regard it in Landry's paralysis as indicative of anything more than the toxic state undoubtedly present.

"4. There was no definite evidence of neuroglial or vessel changes.

"If we subtract from the morbid changes described the diffuse fatty myelin degeneration in the white columns of the cord and in the peripheral nerves, which is common to acute toxic states of various kinds, we have left practically nothing but the ganglion-cell changes in the spinal cord. There is justification, then, for believing that the poison at work in these cases exerted its chief influence on the ganglion cells and that the changes thus induced form the only obvious morbid anatomy of the disease."

As to bacteria, Buzzard found in his first case the following: "1. A micrococcus was isolated in pure culture from the blood of the patient after death. 2. An organism, indistinguishable from that which was cultivated, was found in large numbers in the external part of the spinal theca. 3. A subdural injection of the cultivated coccus into a rabbit produced after some days a rapidly spreading palsy. 4. The same organism was discovered in the theca of the rabbit and isolated in pure culture from its blood. 5. In neither the patient nor the rabbit was the organism to be demonstrated in the spinal cord or the pia-arachnoid, and in neither case were there inflammatory reactions in those tissues. In my second and third cases all efforts to find bacteria, either by culture or by staining of films and sections, were without result. The same lack of success has attended the bacteriologic examination of most cases of Landry's paralysis which have not shown evidence of being in reality cases of inflammatory myelitis."

Differential Diagnosis. The two most important problems are the differentiation of acute poliomyelitis from Landry's paralysis and that of the latter condition from acute toxic polyneuritis. The following points are of differential value:

"1. The *constitutional symptoms* in severe cases of acute poliomyelitis are more marked than those in Landry's paralysis, the range of temperature is higher, often reaching 102°-104° F., the malaise and anorexia more profound, and the vomiting and disturbance of the alimentary canal are more frequent. In children, convulsions are often associated with the former ailment.

"2. *Sensory Phenomena.* In Landry's paralysis there may be, and often is, complaint of pain in the back, of sensations of numbness or tingling in the extremities before and during the onset of paralysis. The only sensation complained of in the paralyzed limb is that of discomfort or of cramps, arising, partly, at any rate, from their immobility; they may be handled without giving rise to anything more than slight tenderness on deep muscular pressure. In acute poliomyelitis the pains complained of are more urgent and affect not only the back, head, and neck, but frequently, the limbs as well. The paresthesias may be present, but a prominent feature is the pain, sometimes very severe, elicited by passive movements of the affected extremities.

"3. *Motor Phenomena.*—In both diseases paralysis is flaccid and may be general. The escape of a single muscle or of a group of muscles in a region where all the others are affected or a marked asymmetry in the condition of corresponding muscles on the two sides of the body are suggestive of acute poliomyelitis rather than of Landry's paralysis. Within a few days or a week of the onset in cases of acute poliomyelitis, if death has not occurred, it is usual to find rapid recovery in some parts and early atrophy and electrical changes in others. In Landry's paralysis recovery, if it takes place, is slow and evenly distributed without marked atrophy.

"4. In both diseases *the sphincters* usually escape, the inability to empty the bladder and rectum, which is often a temporary symptom, being due to the impaired abdominal musculature and the position of the patient.

"Similar general rules for diagnostic guidance can be drawn in the case of Landry's paralysis and acute toxic polyneuritis.

"1. *Constitutional.*—Although the constitutional symptoms in Landry's paralysis may be only of slight severity, it is the rule to find some rise of temperature and associated anorexia. Cases of acute toxic neuritis may, on the other hand, often run their course with no corresponding features.

"2. *Sensory Phenomena.*—In acute toxic polyneuritis numbness, pins and needles, and sharp pains are common in the extremities and very marked tenderness of muscles, sometimes of nerves, is the rule. There may or may not be relative anesthesia in the glove and stocking areas.

"3. *Motor Phenomena.*—These are very important and can only be properly appreciated by carefully and systematically testing the various groups of muscles in the body. In the first place the limbs are more affected than the trunk and the peripheral parts of the limbs more than the proximal in acute toxic polyneuritis. In the same disease, although many of the trunk muscles may be involved, the most marked incidence is generally upon the diaphragm, in which case respiration becomes entirely costal. Of the cranial nerves, those supplying the facial muscles are often picked out and may be the only ones to suffer. Sometimes the palate is also affected. In Landry's paralysis the musculature is affected generally and evenly, the trunk and limbs presenting a degree of paresis which is more or less symmetrical and equal. In cases in which the paralysis first affects the lower extremities it is not uncommon to find these parts most paretic at the time of observation, but the spread of the disease will then be a progressive one in an upward direction and the thoracic muscles may succumb before the diaphragm, or both may be equally weak. When the muscles affected by the cranial nerves are involved deglutition, phonation, articulation, together with the movements of the face and jaws, may be impaired, but the fatal termination generally takes place before any one of these actions is abolished. The condition may be progressive over two, three, or more weeks, and yet be unassociated with any definite local atrophy of muscles, and recovery may take place without its appearance. In acute toxic polyneuritis recovery, even if rapid and good, is usually accompanied by some temporary muscular atrophy and diminished electrical excitability in some of the limb muscles, especially the dorsiflexors of the ankles and the extensors of the wrist and fingers.

"4. *Reflexes.*—The deep reflexes are abolished in both diseases in the affected parts. The abdominal reflexes are often retained in acute toxic polyneuritis and rarely always abolished in Landry's paralysis. The plantar reflexes are absent or flexor in type in both instances and the sphincters are only temporarily, if at all, disturbed. In addition, it is interesting to note that relapses and recurrences occur in toxic polyneuritis, although they are, as far as I know, unrecorded in poliomyelitis or Landry's paralysis."

[A case of relapsing acute poliomyelitis is recorded in this volume, p. 145.—Ed.]

E. Macnamara and J. Bernstein[1] report a case of Landry's paralysis with bacterial findings. The case was clinically a typical instance of Landry's paralysis. The motor symptoms were those of a gradually ascending flaccid palsy, and these mounted to so high a level that on the eleventh day of illness ocular movements were impaired. Thereafter the symptoms gradually retrograded. Throughout there were no sensory symptoms nor any incontinence of feces or urine, and eventually recovery was complete. After reporting their case the authors make elaborate references to the etiology of the disease, particularly to the cases in which bacteria have been reported. They then discuss the symptoms and prognosis, calling attention to the fact that these cases, where recovery does occur and is complete, clearly belong to Landry's paralysis, whereas those in which recovery occurs either partially or very tardily are cases which can only hesitatingly be brought under the heading of Landry's disease. The pathology is also elaborately treated. Bernstein's study of the bacterial examination of the cerebrospinal fluid and of the blood disclosed an organism resembling the tetracoccus isolated from a similar case by Buzzard. Careful descriptions of these studies are appended, together with a very complete bibliography.

E. Stadelmann and M. Lewandowsky[2] also report a case of Landry's disease which resulted in recovery. L. Jacob[3] reports a case combined with hysteria, simulating an ascending spinal cord tumor, ending in recovery. Wharton Sinkler[4] relates a case of Landry's paralysis with recovery. Sinkler inclines to the view that the disease is an infection

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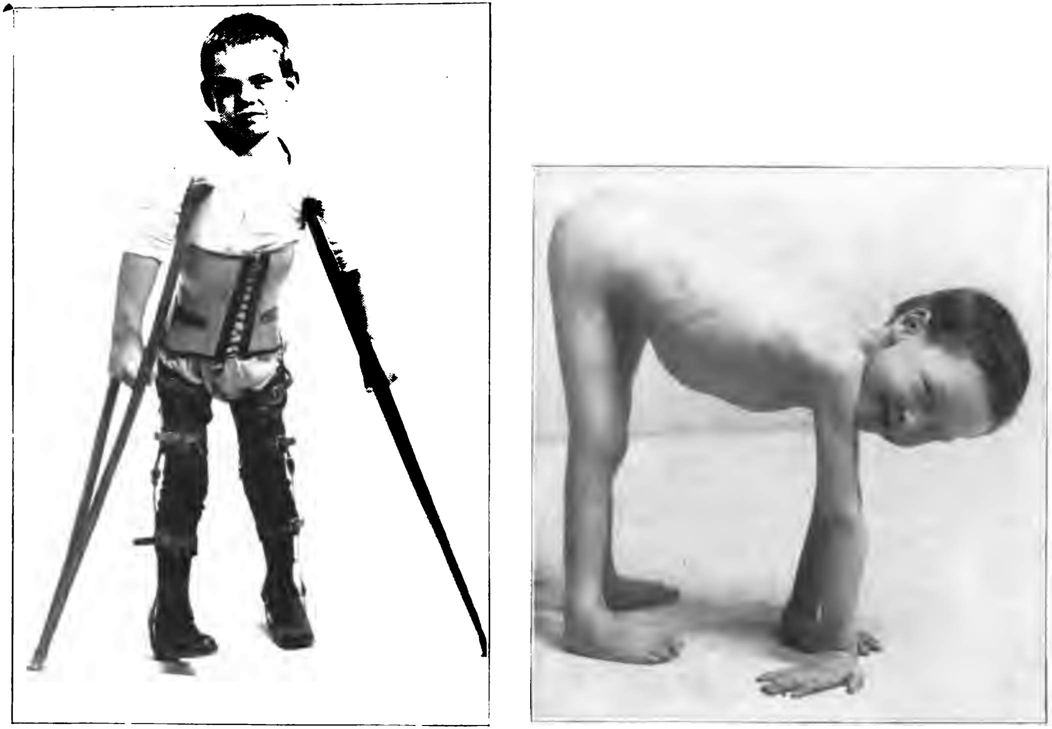
(1) British Med. Jour., Aug. 4, 1906.

(2) Klin.-ther. Woch., Mar. 25, 1907.

(3) Neurolog. Centralblatt, Mar. 16, 1907, p. 264, and April 2, 1907, p. 299.

(4) Jour. Nerv. and Ment. Dis., November, 1906.



A. PLATE V. B.

A. Case shown in Plate IV, after operation. Result.—Ability to assume upright position. Some power of extension of left leg on thigh. Gait active, requiring crutches.

B. Poliomyelitis anterior. Both lower extremities involved. Flexion of thighs. Quadrupedal gait. (Page 146.)

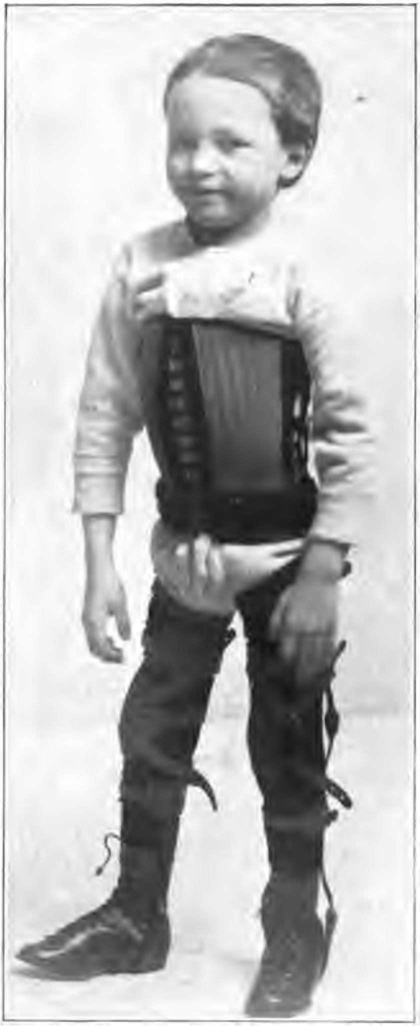


PLATE VI.

Same case as shown in Plate V, B. Treatment.—Arthrodesis of both ankle joints. Transplantation of both sartorius muscles into the paralyzed quadriceps tendons. Division of contracted fasciæ latæ. Apparatus. Result.—Ability to assume the upright position. Gait active, but requires crutch support. (Page 146.)

of microbic origin. Like all cases of Landry's disease which recovered, the patient was completely restored to health.

A case is reported by Mann and Schmaus[1] which showed postmortem a small-celled perivascular infiltration of the anterior horns of the cord. There were no marked evidences either of degeneration of the ganglion cells or of the medullary substance. Unfortunately no bacterial examination of the cord was made; but the cerebrospinal fluid extracted before death was sterile. The conclusion of the authors is that the disease was caused in their case by a toxic substance of unknown origin exerting a harmful influence in the territory of supply of the anterior spinal artery.

Symptomatology. J. N. Hall and S. D. Hopkins[2] report five cases of Landry's paralysis, and discuss its relations to myelitis, poliomyelitis, and multiple neuritis. These authors consider those cases typical which are "characterized by acute onset, motor paralysis, ascending in character, without material involvement of sensation, and with loss of the deep and superficial reflexes, with or without involvement of the sphincters, and generally fatal by respiratory paralysis within one or two weeks, or rarely progressing to a slow recovery." They have collected from the literature of 1905 twenty-three cases by nineteen authors. These are summarized as follows:

(1) Character of muscular paralysis: in legs, ascending, 22; in arms, and ascending, 1. (2) Impairment of sensation: none, 11; not recorded, 2; slightly impaired, 11. (3) Deep reflexes: lost, 13; not recorded, 10; decreased, 1. (4) Superficial reflexes: lost, 11; not recorded, 10; present, 1; decreased, 1. (5) Pain: present, 9; absent, 9; not recorded, 4. (6) Atrophy: present, 1; absent, 14; not recorded, 8. (7) Pulse: above 100, 7; below 100, 3; not recorded, 13. (8) Temperature: normal, 6; moderate, below 102°, 5; high, 3; not recorded, 9. Since 8 out of 23 showed fever, the disease cannot be regarded as generally afebrile. (9) Bladder sphincter: retention, 9; incontinence, 5; not involved, 4; not recorded, 5. (10) Result: recovery, 7; death, 17. Hall and Hopkins are of the opinion that Lan-

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(1) Deut. Archiv für klin. Med., Mar. 15, 1907.
(2) Jour. Am. Med. Assoc., Jan. 12, 1907.

dry's paralysis is due to infections and toxins, which in most cases are bacterial products.

AMYOTROPHIC LATERAL SCLEROSIS.

Differential Diagnosis. R. T. Williamson[1] reports two cases of amyotrophic lateral sclerosis, one of them showing marked bulbar symptoms, and contrasts them with two cases of progressive muscular atrophy. As points of differential diagnosis he instances the following:

1. In favor of amyotrophic lateral sclerosis would be a spastic condition of the legs—spastic gait, scraping of the toes in walking, rigidity of the legs on passive movements, increased knee-jerks, ankle clonus, and the extensor type of plantar reflex, Babinski's reflex.
2. Symptoms of bulbar paralysis would be in favor of amyotrophic lateral sclerosis.
3. Amyotrophic lateral sclerosis, as a rule, runs a more rapid course than progressive muscular atrophy. The duration of the disease in progressive muscular atrophy is often very chronic—ten, fifteen, or twenty years, whilst in amyotrophic lateral sclerosis the disease usually terminates fatally in from one to four years, though a few exceptional cases of longer duration have been recorded.
4. The increase of the wrist-jerks is in favor of amyotrophic lateral sclerosis; loss of this reflex is in favor of progressive muscular atrophy.
5. Paresis or paralysis in muscles which are not yet definitely atrophied is in favor of amyotrophic lateral sclerosis and against progressive muscular atrophy.

FRIEDREICH'S HEREDITARY CEREBELLAR ATAXIA.

Five well-described cases of Friedreich's ataxia occurring in two families, two cases in one, and three in another, are reported by T. W. Griffith.[2] The illustrations show especially well the spinal curvature observed in one case, and the peculiar cocking up of the great toe, with its double curve, so characteristic of these cases. Those in the first family were in two sisters, one 17½ and the other 10½ years of age. The onset of the disease was at the age of 12 years

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(1) Edinburgh Med. Jour., April, 1907.
(2) British Med. Jour., Mar. 9, 1907.

in the older sister, with difficulty in walking. She used to stagger as though intoxicated. The disability continued until she became bed-ridden. The younger sister showed an early stage of the disease. Despite the fact that she presented no pes cavus, skoliosis, nystagmus, or speech disorder, the ataxia and muscular weakness, absent knee-jerks, and the presence of Babinski's toe-sign strongly suggest Friedreich's ataxia. Her elder sister's case practically establishes the diagnosis. The other family, a large one, shows three of its members affected, the elder showing pronounced skoliosis. His feet present a slight degree of inversion, the whole foot appears shortened, the antero-posterior arch is increased, and there is marked dorsal flexion of the metatarso-phalangeal joints with plantar flexion of all the interphalangeal ones.

As to *differential diagnosis,* "Friedreich's ataxia resembles several other affections. Thus it resembles true tabes in the presence of ataxia and absence of knee-jerk. The coexistence of actual muscular weakness, the presence of nystagmus and of speech defects, the curvature of the spine, the deformity of the feet, and the extensor character of the plantar reflex would all be positive signs of Friedreich's ataxia. The tendency for the disease to affect several members of the same family, the early age of onset, the absence of shooting pains or profound sensory phenomena, of visceral crises and trophic lesions, and the comparative immunity of the sphincters would all tell in the same direction, as would also the absence of optic nerve atrophy and of the Argyll-Robertson pupil.

"Gross disease, such as tumor of the cerebellum, may be associated with ataxia and absence of the knee-jerk, but the presence of pain, vomiting, and optic neuritis, as well as the reeling character of the gait, would probably prevent its being confounded with neoplasm.

"In ataxic paraplegia there is, as in Friedreich's disease, a combination of ataxia with weakness, and the plantar reflex is of the extensor character, but the knee-jerk is increased and there may be ankle-clonus; there is no tendency for the disease to affect several members of the same family, and it usually begins at a later age."

Pathology. J. Déjerine and A. Thomas[1] present their pathologic findings in a case of Friedreich's disease. Particular attention was paid to parts not hitherto reported as involved. Until now the lesions have been found in the posterior columns, spreading with less intensity to the lateral tracts, chiefly in the pyramidal, and direct cerebellar tracts, and in the columns of Gowers, or even in the direct pyramidal tracts. The participation on the part of the pyramidal tracts may be considered constant, but that of the other tracts is variable, the direct cerebellar tract being frequently involved, however. The degeneration of the posterior columns scarcely differs from that of tabes dorsalis. Déjerine and Thomas find in their case lesions in the roots, ganglions, and peripheral nerves, following Ramon y Cajal's method by impregnation with silver.

MULTIPLE SCLEROSIS.

Types of Disease W. B. Warrington,[2] in an article on some of the more common nervous diseases in which he places multiple sclerosis as occurring next in frequency after tabes dorsalis, recounts the following types of multiple sclerosis, following Tredgold:

1. The classical type, in which the predominant symptoms are tremor, nystagmus, and alteration of speech. 2. The spastic paraplegic type, in which for a considerable period the clinical picture is indicative only of a lateral sclerosis. 3. The combined lateral and posterior sclerosis type, in which spastic paraplegia is accompanied by sensory change. 4. The transverse myelitis type. 5. The cerebellar type, characterized by the cerebellar gait, and often accompanied by headache, giddiness, vomiting, and symptoms suggestive of cerebellar tumor. 6. The hemiplegic type, in which apoplectiform attacks occur, followed by hemiplegia with or without aphasia. Usually the paralysis is of a transient nature. 7. The hysterical type, marked by transient paresis following nervous shock or actual injury.

Pathology. Takakazu Nambu[3] reports a case of multiple sclerosis from Pribram's clinic at Prague, with post-

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(1) Rev. Neurol., Jan. 30, 1907.
(2) Liverpool Medico-Chir. Jour., July, 1907.
(3) Prager med. Woch., Jan. 17, 1907.

mortem findings. Clinically the case presented the usual symptoms, the onset being a feeling of heaviness in the legs, associated with weakness, so that there was difficulty in walking upstairs. This weakness forced the patient to her bed within a year, and bladder and rectum disturbances followed. In a few months sphincteric control was regained and subsequently she was able to walk again. The illness began in 1894, grew worse steadily till 1898, when the patient began to improve. Six years later, in 1904, the patient was again forced to go to the hospital, where she died, largely from decubitus and cystitis.

A very careful autopsy showed as the most remarkable finding an intense sclerotic process in the spinal cord. In the upper cervical region the sclerosis was diffused through the whole cross-section. Medullary sheaths were found only in the peripheral portion of the right anterior part of the cord, and in the territory of the right anterior root. The ganglion-cells, on the other hand, aside from pigmentary deposits in a few of them, were approximately intact. Free axis cylinders were found only in the right antero-lateral tract, and in both anterior horns. None were found in the lateral tracts or in the dorsal tracts. In the middorsal region the sclerotic patch involved the whole cross-section, and the same was true of the midlumbar region. The relation between the sclerosis and the blood-vessels was everywhere very evident, the changes in them being inflammatory in type.

Symptomatology. Sanger Brown[1] calls attention to the fact already alluded to in the PRACTICAL MEDICAL SERIES, Vol. X., 1905, that multiple sclerosis is seldom characterized by the simultaneous presence of the Charcot tetrad of symptoms, intention tremor, nystagmus, scanning speech, and optic atrophy. It is true that these symptoms separately and variously combined are frequently seen, but they occur only in its later stages, and, as a rule, several years at least, after other symptoms, which if rightly understood, suffice for a diagnosis. Brown describes four cases which illustrate this contention. The first showed "attacks of vertigo, with pains in the eyeballs and the nape for six years; some unsteadiness of gait from the

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(1) Am. Jour. Med. Sciences, Dec., 1906.

first; later diplopia, amblyopia, paresthesia, and weakness of one leg, and sphincter involvement." The second showed "a slowly progressive ataxia and weakness of the legs for six months; then numbness and ataxia in the hands with tonic spasm of the interossei; next diplopia for several weeks; paresthesia of the thighs extending to the feet; girdle sensation about the abdomen; and sphincter involvement." The other two cases need not be summarized. Not one of the four cases showed any anomaly of the fundus oculi. Important in these cases are the facts that motor paresis is early present and usual; that sensory disturbances in the form of paresthesias prevail early; that ataxia is very common; that sphincter involvement is frequent. On the other hand, the so-called cardinal symptoms are in the background. Brown does not agree with Ed Müller[1] that a peculiar affection of the optic nerve, consisting mostly of a simple atrophic discoloration, especially of the temporal part of the papilla, is a most certain and important early sign of multiple sclerosis, or that failure of the abdominal or cremasteric reflexes is a highly diagnostic sign.

Differential Diagnosis. H. Campbell Thomson,[2] after reporting a case of multiple sclerosis, mentions the difficulties of diagnosis, and presents the following summary of differential points:

1. During the early part of the disorder the symptoms may vary so much that there is danger of diagnosing hysteria, or some other functional disease. 2. As the patches of sclerosis develop and become more permanent they are very likely to occur in the course of the crossed pyramidal tracts. In the absence of other signs the diagnosis of spastic paraplegia will be rightly made, but the important question arises as to whether it is to be regarded as primary spastic paraplegia, the prognosis of which is certainly better than that of disseminated sclerosis. 3. The addition of some patches in the posterior columns may cause ataxia, and so give rise to the diagnosis of ataxic paraplegia, and the remarks concerning the fate of cases of seemingly primary lateral sclerosis, apply equally to ataxic paraplegia. 4. When the patches are more freely disseminated,

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(1) Practical Medicine Series, 1905, Vol. X.
(2) Practitioner, April, 1907.

but still do not give rise to the more typical symptoms, the disease may be confused with general paralysis of the insane, Friedreich's disease, cerebellar disease, and, in children, with cerebral diplegia.

Differentiation from Syphilis. William G. Spiller and Carl D. Camp[1] call attention to the clinical resemblance which may exist between cerebrospinal syphilis and multiple sclerosis. The patient, a man 28 years of age, positively denying, during several years, syphilitic infection, presented marked ataxia of gait, intention tremor of the limbs, and, a month or two before death, of the muscles of the face; scanning speech; at first normal iridic reactions, later Argyll-Robertson pupils, with unequal pupils; pallor of the temporal side of the right optic nerve and simple atrophy of the left optic nerve; and vertigo. Pain was never a prominent symptom. Remissions did not occur during the years he was in the hospital. Nystagmus was not observed. Shortly before his death, when some scars on his limbs were discovered and a history of syphilis obtained, the diagnosis of cerebrospinal syphilis was entertained, although it was not forgotten that a patient with multiple sclerosis is not exempt from syphilis.

The lesions found were those of meningo-encephalitis, consisting chiefly of round-cell infiltration and degeneration of the posterior columns of the cord.

The patient was married and was the father of a healthy child. He positively denied having had syphilis until a short time before his death. On the other hand, his occupation, in which he was constantly exposed to the weather, furnished a possible etiology for multiple sclerosis. Pain was never a marked feature, and at no time was of the sharp, shooting character usually associated with inflammatory lesions of the meninges involving the posterior roots. The tremor was of the intention type, and the speech was "scanning." However, there was no nystagmus, yet this is often absent in multiple sclerosis.

Summing up the symptoms in this case it is evident that there was enough or more than enough to make the diagnosis of multiple sclerosis, and that there were no symptoms that were incompatible with such a diagnosis.

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(1) Amer. Jour. Med. Sciences, June, 1907.

Prognosis. Otto Maas[1] reports a case of multiple sclerosis, interesting because of its course. The patient was taken ill at the age of 32 years with acute inflammatory rheumatism, from which she recovered. Six years later, in 1888, she visited an ophthalmologic clinic because of a diminution of vision in the right eye. At that time the right papilla was pale as compared with the left, and there was a doubtful central scotoma. A few months later a numb feeling, together with paresis, appeared in the lower extremities, and there was some retention of urine.

At this time Oppenheim, of Berlin, made the diagnosis of multiple sclerosis, finding in addition to the above symptoms some delay in speech. Slowly the symptoms retrograded so that she was comparatively well. In 1890, however, the eyesight in the *left* eye suddenly began to fail, and a fresh optic neuritis was discovered in it. In the right eye a temporal atrophic excavation was found. In 1892 the weakness of the legs and bladder disturbances reappeared. Again she began to improve, so that in 1893 she was practically well, vision was improved, and the weakness in the legs was entirely gone. For ten years she worked as she used to do, and to all intents and purposes was normal, until in 1903 she was seized with another violent attack of rheumatism, which disabled her until 1905. At present she is again wholly recovered, except that she complains of a certain fatigue in the legs if she uses them for a very long time, and that in coughing or sneezing a drop or two of urine sometimes escapes. The patient is now 56 years of age, having had multiple sclerosis for 18 years. Oppenheim speaks of only three cases of multiple sclerosis in which nothing pathologic could be found from 5 to 10 years later. In no case in the literature has so favorable a course as in this case been recorded, where there has been a remission for thirteen years.

SYRINGOMYELIA.

Pathology. Spiller[2] reports a remarkable case of syringomyelia. Clinically the case presented nothing unknown

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(1) Berlin. klin. Woch., Feb. 18, 1907.
(2) British Med. Jour., Oct. 20, 1906.

in that disease. The implication of nerves of sensation of the head, the paresis of the abducens, and the intense atrophy of the tongue, have been repeatedly observed in this disorder. "The interest in the case lies in the syringobulbia, and the extent of the cavity from the sacral region through the medulla oblongata, right side of the pons, right cerebral peduncle, and right internal capsule, to a point two or three millimeters below the floor of the lateral ventricle in the head of the caudate nucleus. Such an extent of the cavity upward has never previously been observed, and was not suspected during the life of the patient. Interesting also is the situation of the cavity, so that most of the cranial nerves escaped.

"The case further shows that implication of the third, fourth, or motor portion of the fifth cranial nerve does not necessarily imply an associated pathologic process when occurring in syringomyelia, since such implication may be directly due to the involvement of any or all of these nerves in the cavity-formation.

In speaking of syringobulbia, Schlesinger says the cavity does not usually extend above the exit of the glosso-pharyngeal nerve, but that it sometimes reaches the lower part of the nucleus of the seventh nerve. The pons, therefore, forms the termination of even extensive syringobulbia. Hence Schlesinger regards involvement of nerves above the fifth as indicating a complicating lesion, such as hydrocephalus, tabes, or paretic dementia. This case controverts Schlesinger's assertion.

Two cases of syringomyelia are reported by T. K. Monro,[1] together *with an account of the pathologic changes* in the spinal cord of one case by Hugh McLaren. Monro makes the following two classes of cases: 1. In one of these classes there is a condition of gliomatosis. The cord becomes the seat of a new growth which is gliomatous or sarcomatous in its nature, and in which cavities form. A great length of cord may be involved and the organ may be enormously enlarged. 2. In the second class, which is less rare and also less grave, three groups may be recognized. (a) Some are cases of dilatation of the central canal, which may be distended with

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(1) Glasgow Med. Jour., Oct., 1906 .

fluid. Such cavities may have some embryonic tissue around them, or the dilatation may be due to obstruction of the canal by a tumor low down. They constitute *hydromyelia* or *hydrorrhachis interna.* (b) In a second group the cavity seems to have originated apart from the canal, and thereafter to have established a communication with it, in consequence, perhaps, of inflammation or softening. This condition is spoken of as *gliosis,* and is probably of congenital origin. (c) It has been suggested that a third group may be due to minute hemorrhages taking place at birth in connection with difficult or protracted labor.

Symptomatology. The cases reported by Monro show the usual symptomatology. They are interesting in that, combined, they fairly illustrate the entire clinical picture of syringomyelia, the first case showing the early symptoms and the second case the terminal ones. The onset in the first patient was at the age of 21, and consisted of weakness in the right thumb, followed two or three years later by weakness in the right leg, and scoliosis. The sensory disturbance next developed, showing chiefly the characteristic dissociation symptom, and various analgesias, and thermanesthesias.

The onset in the second patient was in his seventeenth year and he died at the age of 38. The initial symptom was a not very painful whitlow, followed in a year by headache, dimness of vision, occasional vomiting, dizziness, falling, and perhaps unconsciousness for a time. Sphincteric disturbances early developed in this patient. The reflexes were variable. For example, the right knee-jerk was exaggerated, and the left could not be obtained, probably due to rigidity of the knee-joint. Ankle-clonus and the toe-sign were bilaterally present. The abdominal, epigastric and cremasteric reflexes were absent. Nystagmus was present. Postmortem examination of the cord at numerous levels showed a cavity "extending horizontally into each posterior horn, and vertically from the second cervical to the middle lumbar region; constituted throughout the greater part of its length by the dilated central canal, but separate from that canal at both its upper and lower extremity."

Anthropathy. L. F. Barker[1] discusses the arthropathies of syringomyelia, which closely resemble those of tabes. About 10 per cent. of syringomyelic cases suffer from joint involvement. Men are about thrice as often affected as women. The average time of onset is the fortieth year, but it may occur in childhood. Usually the arthropathies precede the muscular atrophies, but not constantly so. The joints of one side of the body are as prone to involvement as those on the other, and the shoulders and elbows are most often affected (80 per cent. of the cases). Even the wrist appears to be twice as often attacked as either the hip or ankle.

Trauma frequently precedes the arthropathy. The swelling is usually sudden, as in the tabetic joint lesion. As a rule the joints are not wholly painless. "After a few days the swelling subsides, but some crepitation remains in the joint. In the course of time this subsidence of swelling is followed in turn by a gradual increase of swelling. It may be years before this leads to marked enlargement and deformity of the joint." The diagnosis is made certain by finding muscular atrophy, with the dissociation of sensation.

Differential Diagnosis. L. Alquier and Lhermite[2] call attention to the similarity occasionally observed between syringomyelia and Pott's disease, showing that a differential diagnosis may sometimes be necessary. Both clinically and anatomically these diseases may closely resemble one another. Clinically, for example, the dissociation symptom so characteristic of syringomyelia may occur in Pott's disease. Cervical Pott's disease may simulate syringomyelia when to the spastic paraplegia of the lower extremities is added a radicular paralysis with symptomatology of the upper limbs, and when the dissociation symptom is present. The diagnosis must rest on a search for root-signs, which may be lacking, upon the existence of early root-pains in Pott's disease, the absence of trophic disturbances, the presence of disturbances of the sphincters. These, if present, indicate Pott's disease. Anatomically, cavities like those of syringomyelia in a general way have been found in Pott's disease.

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(1) Jour. Am. Med. Assoc., Feb. 2, 1907.
(2) Rev. Neurol., Dec. 30, 1906.

Treatment. In 1905 Raymond proposed radiotherapy for cases of syringomyelia, and the results were good. Three other cases have since been treated, of which one has drifted from sight, and the other two have been much improved. E. Beaujard and J. Lhermite[1] now report a case. The hypesthesia was diminished markedly in extent, whereas the analgesia and thermanesthesia were but slightly affected. Later these defects also largely cleared up. Even the motor functions were helped.

TUMORS OF THE SPINAL CORD.

Diagnosis. Frederick E. Batten[2] writes of the diagnosis of tumors of the spinal cord. The diagnosis "is a matter of no difficulty in the later stages of the disease when all the characteristic symptoms—pain, gradual loss of power, and impairment of sensation—are present. In the earliest stage of the disease it is quite otherwise; pain is, or may be, the only symptom, and it may be impossible to detect, even by the most careful examination, any impairment of movement or of sensation.

Symptoms. "Tumors of the spinal cord give rise to three definite sets of symptoms: (1) pain; (2) loss of power; and (3) loss of sensation. The relative degrees in which these three symptoms are present depend partly on the situation of the tumor, and partly on the nature of the growth. The tumor may be situated outside the theca, within the theca, or within the spinal cord; the tumors in the first two situations commonly give rise to a considerable amount of pain, the latter to but little pain, although numerous exceptions to this rule are met with."

The three cardinal symptoms are: "*Pain,* which begins gradually, becoming accentuated on movement, on bending down, or on bending the head forward, and radiates down the legs or arms. Associated with this pain there may be actual tenderness on pressure on the spine, and this may be so marked as to give rise to the possibility of caries of the vertebræ being present. 'Girdle' pain may be present and may indicate the level of the lesion, but pain as a symptom does not often lend any special aid in the diagnosis of the position of the lesion. Local tenderness may, however, be of localizing value.

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(1) Semaine Méd., April 24, 1907.
(2) Lancet, Jan. 19, 1907.

"*Loss of power* in the legs is a complaint common to many diseases. When, however, on examination it is found that the loss of power is confined to, or at least affects, certain groups of muscles to a far greater extent than others, and when there is also a history of the gradual onset and increase of this weakness, the condition is highly suggestive of the gradual increase of pressure on the spinal cord or nerve roots.

"*Loss of sensation* probably furnishes the best localizing sign of the lesion. The examination of the patient for the appreciation of the various forms of sensation, tactile, heat and cold, and pain, is important.

"The localizing value of anesthesia and analgesia in the distribution of a root area is of great importance. Pain, loss of power, and loss of sensation are the three chief symptoms; the condition of the reflexes and sphincters varies according to the situation of the tumor.

"The importance of early diagnosis in cases of spinal tumor is great, since from delay irreparable damage may be done to the spinal cord by the prolonged pressure of the tumor. So long as the spastic condition remains this is evidence of conduction through the cord at the seat of the lesion, but if a flaccid paralysis supervenes on a spastic condition it indicates that a physiologically complete transverse lesion of the cord has taken place, and unless rapidly relieved will result in a complete anatomic transverse lesion, from which there can be no hope of recovery.

Treatment. In cases in which there is definite specific infection, mercurial inunction should be pushed to salivation. Even in these cases, if a flaccid paralysis should supervene on a spastic condition, no time should be lost in opening the spinal canal and relieving pressure on the cord. In other cases iodid of potassium should be given in large doses. In the majority of cases, operation is indicated, and should be performed at the earliest moment compatible with accurate diagnosis. The seat of operation should be at the highest level indicated by any symptom."

UNUSUAL TUMORS.

Tuberculoma. Alfred Gordon[1] reports a case of tuberculoma of the spinal meninges, interesting chiefly from the point of view of the patellar reflexes. The patient, a colored girl with well marked pulmonary tuberculosis, lived only two days after entering the hospital. Her initial symptom, six months prior to her admission, was weakness and pain in her right arm. "Two weeks later the same condition developed in the left arm and shoulder.

"A month later, weakness *without pain* appeared in the lower extremities; this weakness gradually increased, so that within a month there was complete paralysis of the legs and right arm. At the same time there was loss of control of the bladder and rectum. Upon examination there was found some preservation of power in the left arm, but the other three extremities were totally paralyzed. Wasting of the entire musculature of the body, and especially of the extremities, was quite pronounced. The paralysis of the legs was flaccid.

"The knee-jerks and Achilles-jerks were abolished; there was no ankle-clonus, no Babinski sign. Test for sensations revealed that pain and touch sense were abolished in both legs as far up as the hips; the rest of the body presented normal sensations. There were multiple bed-sores. The pupils were unequal, the left being the larger; they reacted sluggishly to light and accommodation."

The postmortem examination showed a large cheesy-like mass extending from the fifth cervical to the second dorsal vertebra on the right anterolateral surface of the dura. Light, softened areas were found in the posterior column of the cord and in the white matter between and in front of the anterior cornua. The cornua themselves were deformed, very much diminished in size, with marked degenerative changes in their cells. The author calls attention to the following points of interest:

"1. Pain was only slight through the entire course of the disease until admission to the hospital, when it had wholly disappeared. The rule is that, in meningeal tumors, pain is usually of great severity. 2. The tumor, being

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(1) Amer. Jour. Med. Sciences, May, 1907.

extradural, places the case among the infrequent ones. Schlesinger, who made a statistical study of 400 tumors of the spinal cord, found only two extradural ones. 3. The tumor occupied the lower cervical portion, while the usual seat is the dorsal region. 4. The extension of the pathologic process could be traced through the dura- and pia-arachnoid into the cord. 5. The condition of the reflexes is noteworthy. The rule is that sudden severing of the cord, or sudden compression, will lead to an immediate disappearance of the knee-jerks, which later will be reestablished with signs of spasticity. On the contrary, in chronic cases, with slow and progressive destruction of a segment of the cord, spastic paralysis appears at the onset and remains as such until the end. Yet the above case seems to offer an exception to the above rule in regard to the knee-jerks. The patient presented flaccidity and absent knee-jerks during the entire course of her affection, yet six months elapsed between the onset of the affection and her death,—a period of time more than is necessary for a descending lesion of the lateral columns to produce a spastic state of paralysis." [The fact that the posterior columns showed degeneration may account for the absent knee-jerks.—Ed.]

Primary Melanotic Sarcoma. A case of primary melanotic sarcoma of the spinal meninges is reported by C. Thorel[1] because of the extraordinary rarity of melanotic tumors of the central nervous system and the traditional opinion that, if they do occur, such tumors must be metastatic, since they originate only from pigmented localities, such as the skin, certain mucous membranes, etc. Yet the author calls attention to the fact that pigment has been found on the ventral surface of the medulla and pons, in the region of the olfactory bulbs, and in the Sylvian fissures.

The patient was a man of 43 years of age, and showed the symptoms of a compression myelitis with rectal and bladder paralysis, paresis of both lower extremities, and severe shooting pains in them. At the postmortem examination the melanotic sarcoma was found, but nowhere else in the whole body could the slightest traces of a

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(1) Münch. med. Woch., April 9, 1907.

melanotic tissue growth be found. A similar case was reported by Virchow where the pigmented spots were found on the surface of the right hemisphere and on the base; one was reported by Stoerk, and one by Sternberg.

DIAGNOSIS AND TREATMENT.

Friedrich Schultze,[1] of Bonn, discusses the diagnosis and operative treatment of tumors of the spinal meninges, first reporting two typical cases which illustrate one point needing emphasis, namely, that a preliminary neuralgic stage is not necessarily present in these cases; indeed, there may be no pain at all. The first case reported was that of a young school-teacher, who before consulting Schultze had suffered for six months with weakness and a feeling of numbness in both legs, both having grown much worse in the last six weeks.

Never had there been pain. There was no nystagmus, intention tremor or ataxia of the upper extremities. The abdominal reflexes were lacking, the knee-jerks lively, ankle-clonus was present on both sides, but the toe-sign was wanting. The plantar reflexes were absent. The sensory disturbances extended as high as the 8th dorsal segment.

The arms were free from any symptoms, the fundi were normal, the cerebral nerves intact, the spinal column well formed, normally movable, not painful on pressure anywhere. Schultze's diagnosis hovered between multiple sclerosis and meningeal tumor. The course of the case strongly suggested the former, there being no caries of the vertebræ, and the later symptoms, predominating on the left side, suggested its location. It was found at the operation, and proved to be an extradural fibroma. Recovery rapidly followed after the operation. The second case was in a man, 56 years of age, who was also operated upon, but cystitis followed and death was the ultimate outcome.

Schultze has tabulated all of his supposed cases of tumors of the spinal cord up to the present time, excluding those cases showing malignant metastases. There are in all 18 of them.

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(1) Münch. med. Woch., July 9, 1907.

He believes that the correct diagnosis can usually be made, and sometimes rather easily, since many cases run a typical course. He also believes that an operation in the majority of cases is not dangerous when done over the dorsal part of the cord, especially if the tumor is extradural and the dura is not opened. In almost all the 18 cases an extramedullary tumor was supposed, with great probability, to be present.

Of the 18 cases, three were not operated on; in two no tumor was found (the diagnosis being right, therefore, in 16 cases). In the three not operated on the tumor was found postmortem. Of the 13 cases remaining, six were completely cured and one was permanently improved, making a percentage of recovery above 50 per cent.

As an addendum, written by the author after reading the proof of this paper, he reports a nineteenth case of tumor of the cord localized at the third dorsal segment, and removed by Garré. It proved to be a psammoma between the dura and pia. In this case, too, pain of every sort was lacking; there were merely unpleasant sensations in the back, and a moderate sensitiveness to pressure over the site of the tumor. Improvement set in immediately after the operation.

DISEASES OF THE PERIPHERAL NERVES.

CRANIAL NERVES.

TRIGEMINAL NERVE.

Herpes Ophthalmicus. J. Herbert Parsons[1] reports four cases of acute herpes ophthalmicus. Almost invariably the history is that of sudden onset, with swelling and redness of the affected parts and well-marked delimitation from the normal skin around. "The presence of vesicles is equivocal, but the exact distribution in areas supplied by one or more branches of the ophthalmic nerve and the severe neuralgic character of the pain experienced facilitate diagnosis. The branches of the fifth nerve most commonly involved are the supraorbital, supratrochlear, and nasal. Specially noteworthy is the absolutely unilateral limita-

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(1) Lancet, May 25, 1907.

tion. The branches of the ophthalmic nerve which supply sensory filaments to the globe are by no means always attacked, so that in a number of cases the cornea escapes. The patient should be warned that the blebs will leave indelible scars. These characteristic, slightly depressed white cicatrices, with their typical distribution, afford permanent evidence of a previous attack of ophthalmic herpes.

Trigemial Neuralgia. *Classification.* Fernand Levy[1] discusses at length the etiology, symptomatology, and classification of trifacial neuralgia. In general he divides it into (I) a major and (II) a minor type, subdividing the latter into the dental, malarial, syphilitic, tubercular, infectious (rare), and toxic groups. A (III) great division is neuralgia of the facial plexus, the ordinary *tic douloureux.* The discussion of the symptomatology of each of the forms is taken up in detail.

Treatment. Hugh T. Patrick[1] advocates treatment of trigeminal neuralgia by the deep injection of alcohol into the nerve trunk. A few years ago Schlösser, of Munich, proposed and carried out such deep injections, his technic subsequently being improved by Ostwald, and also by Lévy and Baudouin. The method is as follows:

"The substance used is alcohol, beginning with a strength of about 70 per cent. and gradually increasing it to 90 per cent. To this is added a little cocain or stovain to obviate any immediate pain or discomfort caused by the injection. The instrument used is a needle of large caliber, about 12 cm. long, marked off in centimeters up to five, beginning at the point. The needle is fitted with a stylet of such a length that when pushed home the end is flush with the point of the needle. (See Fig. 5, which is

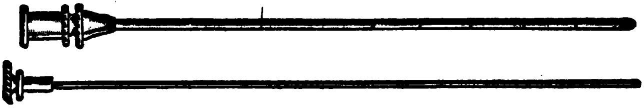

Fig. 5.—Needle and stylet used in the treatment of trigeminal neuralgia. Two-thirds real size.

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(1) Gaz. des Hôpit., July 28, 1906.
(2) Ill. Med. Journal, April, 1907.

about two-thirds size of needle.) For the injection a syringe holding 2 c.c. is employed, the nozzle of which will fit snugly into the end of the needle.

"For the inferior maxillary branch of the fifth nerve the needle is inserted at the lower border of the zygoma, 2½ cm. in front of the anterior border of the external auditory meatus. The bony ridge of the temporal bone, as well as the descending root of the zygoma, can always be felt along the front border of the meatus. The stylet is partly withdrawn, the point of the needle pushed through the skin and subcutaneous tissue, and the stylet is then pushed home, so that for the remainder of the penetration the blunt end of the stylet serves instead of the sharp point of the needle. This is to prevent the possible transfixion of blood-vessels. The needle is then pushed inwards in a plane at right angles to the side of the face and inclined a little backwards to a depth of 4 cm. At that depth the point should reach the inferior branch of the fifth nerve as it emerges from the foramen ovale. The stylet is then withdrawn, the syringe fitted to the needle, and the injection slowly made.

"For the superior maxillary division of the fifth, the point of entrance is at the lower border of the zygoma, ½ cm. posterior to the point which would be reached by prolonging downward the line of the posterior border of the perpendicular process of the malar bone. From this point the needle is passed almost perpendicularly inward, being inclined slightly forward and upward. At a depth of 5 cm. the point should reach the nerve at its emergence from the foramen rotundum into the sphenomaxillary fossa."

Concerning the future of this treatment, Patrick is unwilling to hazard an opinion. "It does not seem reasonable to suppose that injections of alcohol will radically cure trifacial neuralgia. That these injections will hold the disease in abeyance up to a year or more is certain. The operation is practically devoid of danger, requires no anesthetic, and is not difficult to learn. In the great majority of cases it affords immediate relief, and the patient may go about his business within a few minutes after the injection is made.

In a later paper Hugh T. Patrick[1] reports 16 cases treated by this method. His solution is the following:

℞	Cocain hydrochlorate	gr. i
	Chloroform	m. x
	Alcohol	ʒ iii
	Distilled water, sufficient to make	℥ ss

Mix.

Of this he injects 2 c.c. For succeeding injections the proportion of alcohol is increased gradually up to 90 per cent., the amount of cocain being also increased. The results have been most gratifying.

Louis Hauck,[2] who learned the method by seeing Schlösser apply it in his clinic, uses 80 per cent. alcohol, first injecting it into the nerve roots as they emerge from the skull, and, when this does not suffice, injecting it at the foramina, through which the nerves reach the face. "The point of entrance for injecting the first and second branches at their roots is just below the malar bone and in front of the inferior maxillary bone; and for the third branch, below the inferior maxillary bone, about one-half to three-quarters of an inch in front of the angle." . . . The needles used are especially made, the length just right for the different sites. "The swelling often following the injections is treated by hot, wet applications." Three cases are reported by Hauck, all successful.

Brissaud, Sicard and Tanon[3] report 18 cases of trigeminal neuralgia treated by the injection of alcohol, following the method of Lévy and Baudouin. They have, however, discarded the painful trocar of Lévy and use a simple platinum needle of capillary diameter (0.7 of a millimeter, outside diameter, and 0.3 to 0.4 of a millimeter, inside diameter), and 5 to 6 cm. in length. They inject 1.5 c.c. of 95 per cent. alcohol, with or without stovain. They have had no accident of any sort. As a rule, pain quickly subsides and relief lasts, on an average, from three to five months. Recurrences are easily treated by additional punctures.

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(1) Jour. Am. Med. Assoc., Nov. 9, 1907.
(2) St. Louis Med. Review, May 11, 1907.
(3) Revue Neurolog., Mar. 30, 1907.

Schlösser[1] reports 123 cases of trigeminal neuralgia treated by injection of alcohol of a strength of 70 to 80 per cent. The duration of cessation of pain averaged 10.2 months.

Alexis V. Moschcowitz[2] advocates *surgical treatment* of trigeminal neuralgia, by a special method of his own. Concerning the Hartley-Krause operation, he affirms that the mortality is high, and recurrences occur even in competent hands, due to regeneration and reunion of divided nerves. The author then introduces a new principle, calculated to prevent mechanically any regeneration of nerve-trunks and reunion with peripheral portions. This consists in plugging the canal or foramen transmitting the nerve. In his first case he used silver foil, hammering it in tightly. Subsequently the author states that an article by Perthes[3] came to his notice. In this article was recorded an experiment upon a dog in which both infra-orbital nerves were evulsed, and then *one* foramen was plugged with gold. Seven months later the dog was killed, and on the unplugged side was found a newly formed, complete nerve; while on the plugged side there was no union between the divided ends of the nerve.

In a second article[4] the author elaborates his method of radical cure. He says: "Messrs. Tieman & Co. have prepared for me silver buttons, each consisting of a flat top not more than one-sixty-fourth of an inch in thickness, and of a diameter of about one-quarter of an inch, sufficiently firm to retain its shape, and yet sufficiently malleable so that it will give if lightly hammered upon. In its center there is annealed a silver rivet, ranging in thickness from one-thirty-second of one inch to one-eighth of one inch, and about three-eighths of one inch in length. These buttons are shown in the accompanying illustration.

"The operation consists in the following steps: Through an incision the foramen in question is exposed. The nerve is then isolated from the accompanying vessels; a short length is drawn out of the foramen and divided. The accompanying vessels are treated in a similar manner, after double ligature. A button of suitable diameter is selected and driven into the foramen, so as to fill it completely. As some foramina are not absolutely round, the overhanging flat top is now moulded to the underlying bone, so as to make the closure absolutely tight. The small cutaneous incision is closed with fine silk."

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[1] Wien. med. Woch., June 15, 1907.
[2] Med. Record, Sept. 29, 1906.
[3] Deut. Zeit. für Chirurgie, Vol. 77, p. 401.
[4] Med. Record, Feb. 16, 1907.

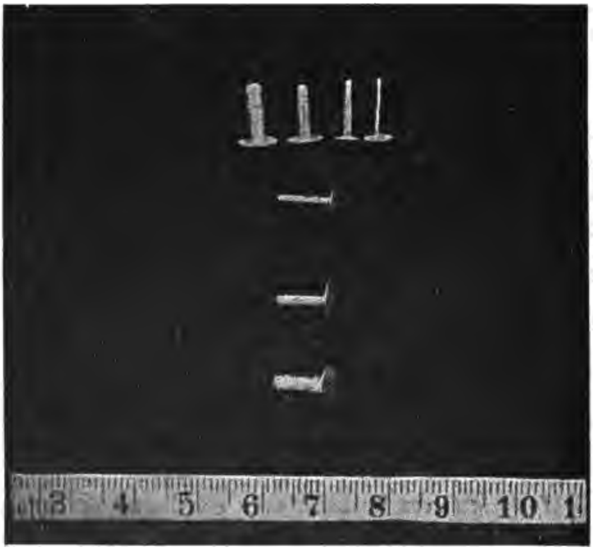

Fig. 6.—Silver buttons used in the treatment of trigeminal neuralgia.

FACIAL NERVE.

Bell's Palsy. L. Pierce Clark[1] makes a contribution to the pathology of refrigeration facial palsy (Bell's facial paralysis). He affirms that up to the present time no cases of refrigeration (Bell's) palsy have been studied pathologically. Indeed only three cases figure extensively in the literature—those of Minkowski (1891), Déjerine and Theohari, cited by Minkowski in the report of his case, and Alexander (1901). The findings in all are affirmed by Clark to be quite unreliable, "as the examinations were made in all three some fourteen years ago, at

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(1) Amer. Jour. of the Med. Sciences, May, 1907.

a time when the distinction between the histological changes of inflammation and degeneration of peripheral nerves were not so definite as they are today." Clark's cases of Bell's palsy were treated by faciohypoglossal anastomosis, four months and twelve years, respectively, after the onset of paralysis. At each operation a piece of the facial trunk was removed for microscopic study. In the first case the nerve-trunk was in a state of incomplete degeneration. There was marked invasion of the tissues with leucocytes, and the neurilemma cells were shrunken and shriveled. The perineural sheath was markedly infiltrated with leucocytes. In the second case the nerve fibers were completely degenerated, showing but occasional granules belonging to the myelin sheath. There were no undegenerated fibers. From both the pathologic and clinical evidence, facial paralysis is a degeneration, and not a neuritis. The clinical evidence that the palsy is not a neuritis consists of the infrequency of pain, absence of tenderness on pressure, and of soreness on passive movements.

Herpes Auricularis. J. Ramsay Hunt,[1] in studying herpetic inflammation of the external auditory meatus, has come upon a new syndrome which he fairly definitely establishes. In brief, he shows that the seventh cranial nerve (called a pure motor nerve in all text-books of anatomy) is a mixed nerve, like the trigeminal. It has a sensory ganglion which is the geniculate ganglion. The motor root of the geniculate ganglion is the facial nerve, and the sensory root is the nerve of Wrisberg, the pars intermedia. Below the geniculate ganglion the peripheral divisions of the facial nerve are found, the great and lesser superficial petrosal nerves, the external petrosal, and the chorda tympani. The geniculate ganglion is of the spinal ganglion type, and therefore in common with such ganglia comes within the realms of pure herpes. The herpetic zone for the geniculate ganglion is found in the interior of the auricle, and in the external auditory canal. Moreover, the geniculate ganglion, being a sensory one, neuralgias may occur, and actually do, under the form of herpetic otalgias. Hunt reports a case of herpes facialis

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(1) Jour. of Nerv. and Ment. Dis., February, 1907.

and occipitocervicalis with facial paralysis of short duration, which seems to bear out his conclusions.

Facial Spasm. Lévy[1] says that facial spasm is of peripheral origin, and is not a reflex affair. This is indicated clinically by the limitation, at the beginning of the spasm, to a few muscle fibers only, and its gradual extension throughout a whole muscle, and then to other muscles. The fact, too, that these cases can be cured by injection of alcohol, constitutes a new proof of their peripheral origin. Schlösser[2] has treated eleven such cases of facial spasm by alcoholic injections, with nine recurrences.

AUDITORY NERVE.

What the author calls *polyneuritis cerebralis acuta,* with involvement of the auditory nerve, and what Frankl-Hochwart calls *polyneuritis cerebralis menieriformis,* is reported by S. Schoenborn.[3] It seems to happen that in multiple neuritis a few individual cases occur in which the cranial nerves alone are involved. Yet not quite 20 cases of polyneuritis cerebralis acuta in which the auditory nerve is involved have been reported. The patient showed an affection beginning acutely, but not in an apoplectiform manner, involving the left abducens, left facial, and both auditory nerves, the right less than the left. Tumor cerebri and meningitis were ruled out by the course of the case. Basal hemorrhage was not regarded likely because the onset was not sufficiently abrupt. The symptoms on the part of the eighth nerve were noises in the ears, vertigo and deafness, with signs of a neuritis in other cranial nerves, in the seventh and the sensory portion of the fifth. Frankl-Hochwart supposes these cases to be upon an infectious basis. Unusual is the involvement of the abducens, which was affected in this case. By the patients the affection is attributed to a cold, but it seems to be toxic in origin. In favor of cold as an etiologic factor is the presence of labial herpes and fever in several of the cases. No Menière-symptoms occurred in the case reported. Vertigo and deafness were present, but there were no seizures as in Menière's disease.

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(1) La Semaine Médicale, Aug. 8, 1906.
(2) Wien. med. Woch., June 15, 1907.
(3) Muench med. Woch., May 14, 1907.

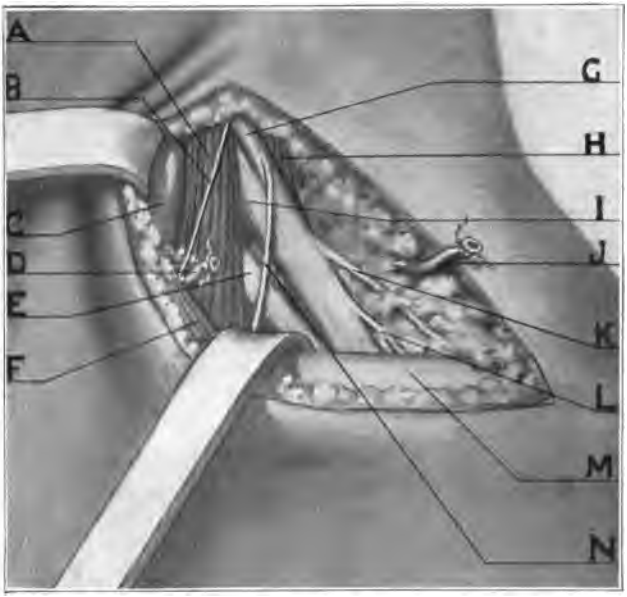

PLATE VII.

Operation for relief of brachial paralysis. A, scalenus anticus muscle. B, phrenic nerve. C, internal jugular vein. D, transversalis colli artery. E, seventh root. F, omohyoid muscle. G, fifth root. H, scalenus medius muscle. I, sixth root. J, transversalis colli artery. K, suprascapular nerve. L, external anterior thoracic nerve. M, clavicle. N, nerve to subclavius muscle. (Page 186.)

PNEUMOGASTRIC NERVE.

Vagal and vasovagal attacks form the subject of an interesting lecture by Sir William Gowers,[1] and 11 illustrative cases are reported, seven of which were not related to epilepsy, and four of which were so related. The term "vagal attacks" is a useful designation for prolonged seizures, the symptoms of which consist chiefly of disturbance of some of the functions of the pneumogastric nerve, for the most part sensory and objective. "With the vagal symptoms there is often combined a slight mental change, and also disturbance of the vasomotor center, causing constriction of the vessels, and coldness, especially of the extremities. Associated with the latter may be some sensory impairment, and sometimes also a form of slight tetanoid spasm. When the vasomotor spasm preponderates the case may be termed 'vasovagal.' The attacks are never really brief; they seldom last less than ten minutes, and more often continue for half an hour or more. There is a sudden onset of slight symptoms, rapidly increasing, and the ending is gradual. The seizures recur at varying intervals, often for months or years. Women suffer far more frequently from these attacks, but they are also met with in men. This, and the fact that the pneumogastric and vasomotor systems are readily influenced by emotion, has probably led to the submergence of these attacks beneath the vague conception of hysteria."

The vagal symptoms are chiefly referred to the stomach, respiratory system, and heart. We may probably ascribe to the gastric nerves a sensation referred to the epigastrium, beginning suddenly, irrespective of the state of the stomach or its functions, and often seeming to ascend to the chest, seldom to the throat or head. "There is seldom nausea, never vomiting. Even more common, especially as an early symptom, is a sense of respiratory distress, of difficulty in breathing. It is sometimes so intense as to amount to orthopnœa and to compel the sufferer, if lying, to sit upright. With this may be combined cardiac symptoms, discomfort, acute pain in some cases, often a sensation of sudden stoppage of the heart, followed by rapid action. With the dyspnea, or the car-

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(1) The Lancet, June 8, 1907.

diac sensation, or both, is often associated a sense of impending death, so intense that no recollection of its falsity in preceding attacks prevents the conviction of its present reality. Although there is no impairment of consciousness, a peculiar slight mental state is common, and may be the first symptom. It is generally described as a difficulty or slowness of mental operations, a difficulty in thinking or in concentrating attention. The vasomotor spasm sometimes attains a high degree. To it the symmetrical coldness is due, for the pulse becomes small at the same time. When general, there is pallor of the face. Shivering is common and may amount to a definite rigor. With the coldness of the extremities, tingling and numbness in them are often described, and sometimes there is tetanoid spasm.

"In the *treatment* of sufferers from such attacks it is essential to discover any defect in the general health and anything that involves strain on the nervous system, and to put these right as far as may be possible. Conspicuous anemia is not often found, but constipation is common and harmful, especially with its attendant indigestion. It is very seldom that any exciting cause can be traced for the attacks that can be avoided, but it is important that the patient should be free from all causes of overfatigue. When an epileptic element can be traced, much good is usually obtained from bromid in moderate doses, combined with belladonna or digitalis. Nitrit of amyl deserves a trial at the onset of attacks, and a slight inhalation of chloroform if the opportunity presents itself. But in all cases in which there is vasomotor spasm the regular administration of nitroglycerin is by far the most useful agent. Taken regularly, although no effect may be perceived from it at the time, it seems to exert a permanent steadying effect on the vasomotor center, and strychnin may be combined with it with advantage."

HYPOGLOSSAL NERVE.

Hemilingual atrophy from fracture of the base of the skull and injury to the hypoglossal nerve where it passes through the anterior condyloid foramen is reported by

Smith Ely Jelliffe.[1] Four previous instances of such injury to the hypoglossal are on record. A Roentgen photograph shows a distinctly anomalous shadow in the region of the third cervical vertebra, but is not clear enough to be positive evidence of injury at that point.

SPINAL NERVES.

Pathogenesis of Neuritis. Lesions of the spinal and cranial nerves have been produced experimentally by toxins, by David Orr and R. G. Rows.[2] They found evidence of a continuous flow of lymph upwards along the nerves to the cord. This led them to look for lesions of the posterior columns in the cords of cases in which some septic focus existed, their idea being that such lesions would be occasioned by an ascending flow of toxins in the lymph stream. "On examining cases of brachial neuritis (infective), bed-sores, suppurating knee-joint, and septic psoas abscess, we found in the cord of all the lesions expected. We then submitted our theories to experimental test, and have been successful in inducing posterior column lesions in rabbits exactly similar to those already found in man.

"The method which we adopted consisted in filling a celloidin capsule with a broth culture of a certain organism. The sciatic nerve being exposed, the sealed capsule was placed in apposition with it, after which the gluteal muscles were stitched in their original position and the wound closed. As toxins are known to diffuse through the wall of these capsules, we assumed they would find their way into the ascending lymph stream of the nerve, and so to the cord, where their presence would be demonstrated by the ensuing degeneration." The organisms employed were *Staphylococcus pyogenes aureus, Bacillus pyocyaneus,* Gaertner's bacillus, and the *Bacillus coli.*

The lesions of the posterior columns in the rabbit's cord were very definite. The degeneration commenced where the sensory fibers lost their neurolemma, and spread inwards to the root-entry zone. The collaterals springing from this area and passing into the gray matter were also

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(1) Jour. of Nerv. and Ment. Dis., March, 1907.
(2) Brit. Med. Journal, April 27, 1907.

affected. The appearance of the affected fibers in both transverse and longitudinal sections shows that the degeneration in its early phase is essentially a primary one of the myelin sheath alone. In no case did the spinal roots outside the cord or the sciatic nerves show any change.

The authors draw the following conclusions: "1. Toxins readily travel up spinal and cranial nerves to the central nervous system. 2. While these nerves in their extramedullary portion possess a neurilemma sheath and are protected by its vital action, in their intramedullary part, having lost their neurilemma, they immediately undergo degeneration. The first change is a primary degeneration of the myelin; axis cylinders and nerve cells are affected later.

"The results of our investigations suggest the possible lymphogenous origin of some nervous affections. We know that tabetiform and cranial nerve lesions in general paralysis, and even those in tabes, are a primary affection of the myelin sheath, commencing where the neurilemma is lost. In our clinical cases, and also experimentally, we have found similar lesions starting at the same point, the result of absorption from a definite toxic focus situated outside the central nervous system, the toxins gaining access by the lymph stream. May it not be possible that the former lesions are also the result of toxins passing to the cord, medulla, and pons by the lymph stream from some external but as yet unknown focus?"

BRACHIAL NEURALGIA.

Brachial neuralgia is thoroughly set forth by Charles L. Dana.[1] It is a malady of the upper cords of the brachial plexus and its branches. The pains are therefore mostly in the upper arm and shoulder, rarely and secondarily in the forearm and fingers. The circumflex, musculospiral, musculocutaneous and internal cutaneous nerves are most involved, and, with the exception of the last, come from the fourth to the seventh cervical nerves. "The pains are usually most intense deep in the arm about the shoulder and at the edge of the scapula. There are few darting

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(1) Medical Record, Feb. 23, 1907.

pains along the whole nerve, to the fingers, hence we assume the process is not usually a root neuritis, and not a trunk neuritis, but a *terminal neuritis.* It is only late and in severe types that there is evidence of the large trunks being involved.

The Nature of Pains About the Shoulder. Pains just above the shoulder and about the acromion are due to involvement of the supra-acromial nerves, which are branches of the fourth cervical. The deep-seated pains referred to the parts about the joint and in the deltoid are caused by the circumflex nerve, which originates from the fourth, fifth and sixth cervical, and which is a sensory motor nerve. The superficial circumflex nerve-pains are felt more posteriorly and over the body of the deltoid; there is a painful point where the nerve becomes superficial. (Fig. 7.)

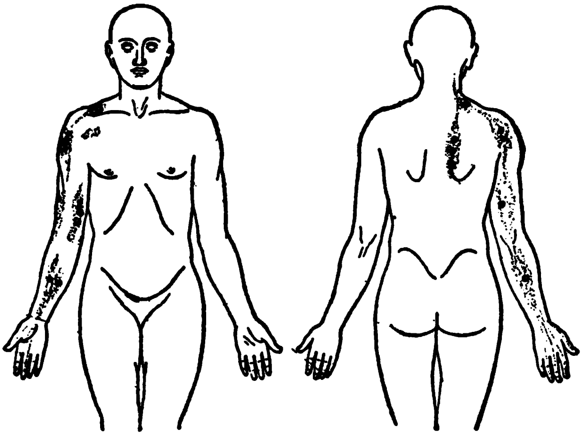

Fig. 7.—Showing painful spots in brachial neuralgia.

Pains on the anterior and inner surfaces of the shoulder and upper arm are due to involvement of the intercosto-humeral, second dorsal. Shoulder pains, however, generally may be said to be circumflex and supra-acromial and to mean involvement of the fibers from the fourth, fifth and sixth cervical nerve-roots.

"*The Nature of Pains in the Upper Arm.* Pains here are generally associated with pains below, in the forearm and hand, and still more with shoulder pains. Usually in brachial neuralgia the pain is most dominant in the arm, often in the upper two-thirds, and rather more on the inner side along the biceps, extending to the axilla. The superficial parts on the inner arm here are supplied by the internal musculocutaneous and internal cutaneous nerve; the deeper parts, anteriorly by the musculocutaneous, which also has a branch to the periosteum and bone and is the seat of the deeper pains. Tender points are felt where the musculocutaneous nerve emerges, and where the lesser internal cutaneous and cutaneous branches of the radial emerge.

"*Pains in the Forearm and Fingers.* There are rarely any neuralgic or neuritic pains confined to the *forearm* alone. Here we have only the ulnar pains following the ulnar nerve, and very characteristic radial pains following the back of the arm to the fingers; and least frequently median nerve pains. Sometimes the whole hand is involved in neuralgic pain, but this is usually hysterical."

Brachial neuralgia and neuritis is, then, a disease of the brachial plexus "characterized by severe pains centering in the upper arm and usually involving the whole upper extremity and shoulder; it runs a course of several months and is due to irritation or inflammation of the trunks of the brachial plexus and its roots of origin. This irritation does not usually reach the degree of neuritis, and the disease does not show the paralysis and atrophy, anesthesia and vasomotor symptoms of neuritis. This form of neuralgia, excluding the cases of traumatic neuritis, is now by no means a rare disease. Among the neuralgias brought to the attention of the neurologist it ranks third, coming after the facial neuralgias and closely following in frequency the sciaticas." Dana believes that brachial neuralgia is increasing, since in 1888, when he collected reports of 453 cases of neuralgia, he found but eight cases of brachial neuralgia, whereas in 197 cases of his own in recent years there are 41 cases of brachial neuralgia. He has also been struck by the fact that brachial neuralgia is seen more frequently in women, especially those of the leisure classes. As to etiology, he finds that the disease occurs oftenest in persons in middle life, ranging from 35 to 50 years of age. In some cases it seems to be due to inordinate use of the arms in simple manual work, and has followed skirt-carrying, a habit prevalent among many women. In some, the use of the arms in dressing the hair may be an exciting cause. There is often a predisposing neurotic or rheumatic taint, and sometimes perhaps an autotoxemia.

The *symptoms* of brachial neuralgia are somewhat as follows: "The onset is rather sudden, but there may be severe preliminary aching in the arm for a few days, and sometimes there are short preliminary or abortive attacks. Usually, however, the patient wakes at night or notices in the morning a severe pain involving the upper arm and shoulder, or perhaps the whole arm. The pain usually is most acute on the inner and front side of the arm, and in the back between the shoulders. It is not a darting, shooting pain, but general and diffuse, involving for a time the whole arm with a pain of paralyzing intensity. The pains exacerbate, coming on at night more severely, or more in the morning, and always after exertion. Movement makes the suffering worse; but the arm can be handled gently without much pain. Some suffering is present nearly all the time, often robbing the patient of sleep; but it may let up for a few hours during the day and then come back again with paroxysms of great intensity, lasting for one or two hours.

When the pain first comes on there are no objective changes in the arm. It looks normal and is simply tender and sore to the touch. Later the arm becomes a little swollen and the fingers somewhat edematous, and there is slight flabbiness of the tissues, showing vasomotor change.

Examination shows points of tenderness along the course of the nerve on the inner side of the arm at the elbow and over the deltoid. There is also a point of tenderness between the scapulæ about at the level of the second or third dorsal vertebra. This is very painful and is characteristic. Stretching out the arm and drawing upon the brachial plexus brings out pain, just as it does in the extension of the hip in sciatica. Later there is also weakness of response to the elbow-jerk, just as in the tendo Achillis in sciatica. The tender points that one finds in brachial neuralgia are by no means always identical with the tender points of Valleix. They vary somewhat, and only correspond in a general way to the distribution of the cutaneous nerves (Fig. 7).

Diagnosis. In chronic and less severe cases the patient is always first treated for rheumatism. It is safe to say that arm pains are never purely rheumatic unless objective evidence presents of myositis, arthritis, or periarthritis. More difficult is it to distinguish between real neuritis of high grade and neuralgia. Neuritis at present is a diagnosis *a la mòde* and pleases the laity, but a neuritis cannot be recognized except by objective signs. There must be pain or tenderness along the nerve, definite sensory losses, muscular weakness, perhaps atrophy and electrical changes. In true brachial neuralgias, objective signs are lacking; for, in a majority of cases, the condition is not one of inflammation, but is an exhaustion neurosis with some rheumatic or metabolic irritation of the nerves in addition.

Treatment. "No treatment of brachial neuralgia is of avail without rest. This must be secured by a sling and, if necessary, by splints. In a good proportion of cases, some relief is gotten by the salicylates, especially aspirin, but they must be given in very large doses, just as for acute rheumatism. Neither massage nor hot baths should be given at first, but after two or three weeks, hot applications of mud or flannel can be used. The patient must understand that, as a rule, it takes ten or twelve treatments to accomplish results. After a course of salicylates, tonics can be given." Of these, the best is strychnin.

G. W. Jacoby,[1] writing of the intradural resection of the posterior roots of a number of spinal nerves for the relief of intractable neuralgia, reports a case in which were resected all of the posterior roots of the brachial plexus. The general impression among surgeons and neurologists is that this operation is to be feared as one in which the results obtained are incommensurate with the risk incurred. The dangers, as shown in the published cases, resolve themselves into a persistent Brown-Séquard paralysis of moderate degree, and two deaths. The Brown-Séquard paralysis was probably due to pressure upon the cord in consequence of the formation of

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(1) N. Y. Med. Journal, Aug. 3, 1907.

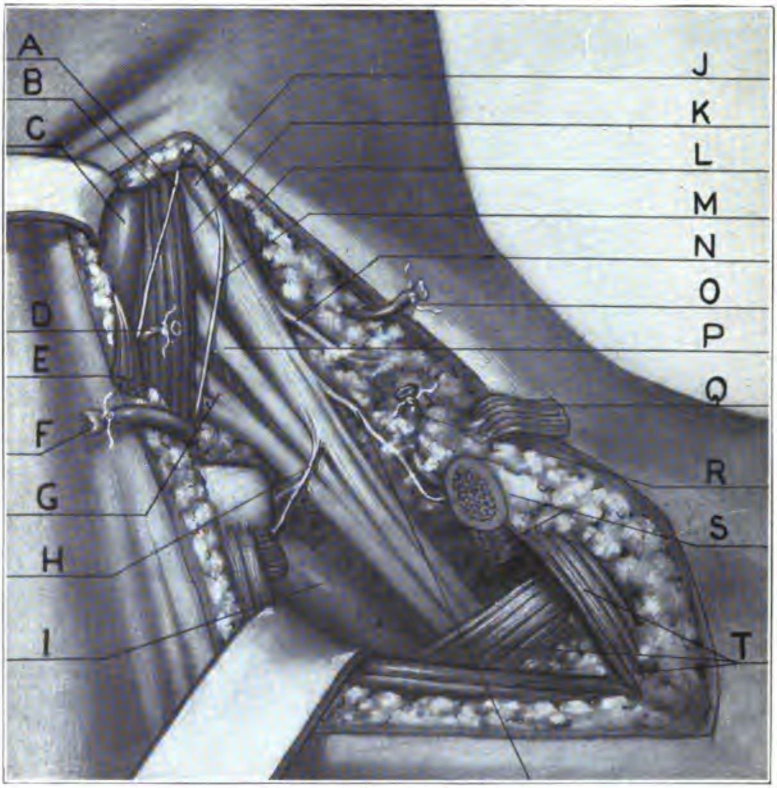

PLATE VIII.

Operation for relief of brachial paralysis. A, phrenic nerve. B, scalenus anticus muscle. C, internal jugular vein. D, transversalis colli artery. E, omohyoid muscle. F, suprascapular artery. G, eighth cervical and first dorsal roots. H, muscular branch. I, subclavian vein. J, fifth root. K, sixth root. L, scalenus medius muscle. M, nerve to subclavian muscle. N, suprascapular nerve. O, transversalis colli artery; omohyoid muscle. R, suprascapular artery. S, clavicle and subclavius muscle. T, pectoralis major, pectoralis minor and deltoid muscles. U, anterior thoracic nerve. (Page 186.)

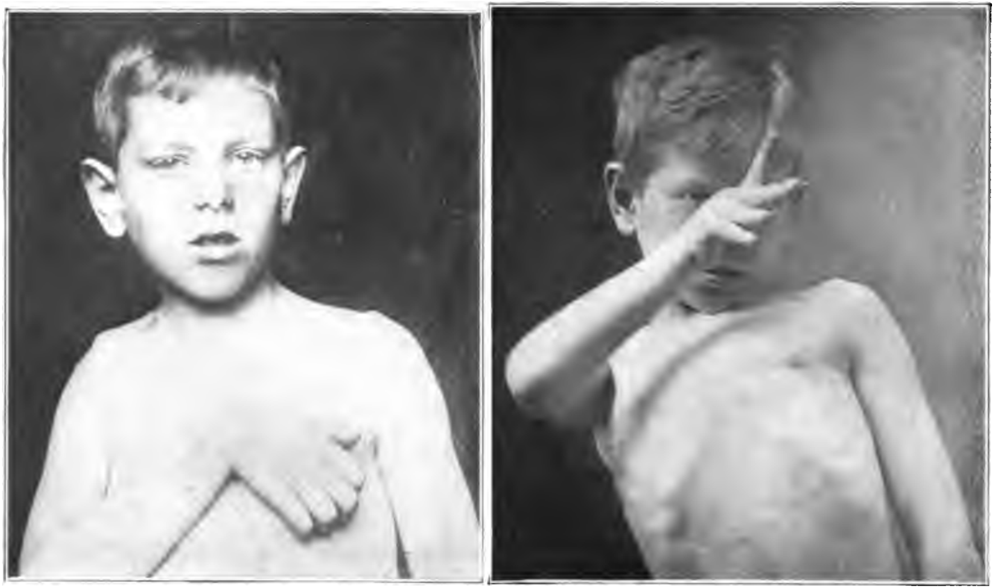

PLATE IX.

A. Before operation. Highest elevation of hand and forearm possible.

B. Ten months after operation, showing increased power of elevation. (Page 186.)

a clot or callus. Certainly the section of the nerve-roots alone bore no part in the causation of the palsy. Jacoby severed the posterior roots of the fifth, sixth, seventh and eighth cervical nerves, and first dorsal nerve within the dura. Besides this case, Jacoby has collected and tabulated fourteen others. A study of the fifteen cases demonstrates that the indications for the operation are: 1. Persistent neuralgias of mixed nerves in which the motor functions of the nerves are intact and in which these are of sufficient importance to warrant their preservation. 2. In neuralgias of radicular origin. 3. In symptomatic neuralgias in which the cause of the pain can be abolished by no other means, and as a palliative measure when the causative affection is irremediable yet pain must be suppressed.

"From this analysis the conclusion seems to be warranted that as an operation for the relief of protracted and intractable pain, the intradural resection of a sufficient number of posterior nerve-roots is one which can be advocated without reserve."

BRACHIAL BIRTH PALSY.

Alfred S. Taylor[1] presents an interesting paper upon the results obtained from the *surgical treatment* of brachial birth palsy. In a former paper by Clark, Taylor and Prout[2] it was shown, as a result of twenty dissections and many experiments on infantile cadavers, that tension (overstretching) of the nerve-roots of the brachial plexus was the essential cause of this lesion. Observations on nine operative cases led to the same conclusion. The roots give way from above downward. The overstretching is caused by pulling the head and shoulder away from each other, as in pulling on the shoulders to deliver the after-coming head in breech cases, or pulling on the head when the shoulders are obstructed in vertex cases. That the above is true was demonstrated by the careful dissection of the brachial plexus of an infant dying two hours after birth, delivered after version, and showing a limp paralysis of the right arm. Notes upon this case by Dr. T. P. Prout are appended to Taylor's article.

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(1) Jour. Am. Med. Assoc., Jan. 12, 1907.
(2) Am. Jour. Med. Sciences, October, 1905.

The accompanying Plates VII and VIII illustrate the technic of the operation. The dissection is rapidly carried through the deep cervical fascia to the brachial plexus, the nerve-ends are found and are brought into apposition by fine silk sutures through the nerve sheath only, while the neck and shoulder are approximated by a bandage to prevent tension on the sutures. Cargile membrane is wrapped about the anastomosis to prevent connective tissue ingrowth. The wound is closed with silk, and a firm sterile dressing applied. The bandage is left on three weeks. A plaster-of-paris dressing is placed on the child before operation and is allowed to harden. At the operation it is removed and trimmed, and replaced when the operation is finished.

Nine cases have been operated on, seven without shock or rise of temperature, primary union following. Improvement in the nutrition of the limb is the first sign of regeneration, just as in facial nerve anastomosis. This change begins at about the end of four months. The return of power is usually rapid, and may be complete at the end of six or eight months, though in one case it was not complete until fifteen months had passed. Systematic after-treatment (massage, electricity, passive motion) is important. Fifteen photographs accompany the article, a study of which demonstrates that the chief handicap to free range of motion lies in the absence of external rotation of the humerus because of the contracted pectoralis major muscle. If, however, the suprascapular nerve is sought out and sutured, this defect disappears. Two photographs are appended as illustrating the results possible. See Plate IX, A and B.

The author's conclusions follow: "Brachial birth palsy of the Erb type is due to overstretching of the nerve-roots, which results in rupture of the sheath, fibers and vessels of the nerves. From the cicatrization of the resulting hemorrhage there results permanent obstruction to the transmission of nerve impulses. The only way to reestablish nerve conduction is to excise the cicatricial area and do an end-to-end suture of the nerves.

"If operation be delayed too long there result: Impaired development of the extremity; contractures of muscles and ligaments, and atypical shape of the joint-ends of the bones, all of which cause the characteristic deformity of this lesion, and which render postoperative return of the extremity toward normal exceedingly slow and laborious. The older a patient is before operation, the slower and more incomplete is the return to normal. In many cases the deformities should be prevented by the systematic use of massage, etc., both before and after operation.

"The damage to the nerve-roots in the order of severity is from above downward. The suprascapular is always damaged, controls the important group of external rotators of the humerus, and should therefore be most carefully sutured to a good proximal nerve stump at operation. Following operation there come improved nutrition and growth of the extremity, with increased range and power of motion. Nerve-bridging may succeed in the very young."

W. B. Warrington[1] records some observations on *paralyses of the brachial plexus.* Excluding the invasion of the roots by diseases of the meninges and vertebral column, and pressure by new growths or aneurism, the common causes are: (1) indirect violence, known, when it occurs in adults, as Erb's or Klumpke's palsy, or, if at birth, as Duchenne's palsy; and (2) rarer cases of neuritis. The general impression is that brachial paralysis occurring at birth is due to direct compression of the nerve trunks between the clavicle and transverse processes of the vertebræ or the first rib. In Warrington's opinion, however, it is due to tearing of the roots of the brachial plexus by the accoucheur, in accordance with the evidence advanced by Clark, Prout, and Taylor.

The symptoms resulting are losses of function of the nerve tissue, and shed no light on the extent of destruction. In mild cases, pressure, the result of inflammation, may destroy conductivity, and the same loss occurs in complete nerve division. We can, however, to some extent decide between complete and partial loss of conductivity. The signs of *complete* loss of function are (a) alteration of sensation, which disappears in time; (b) motor paralysis; and (c) electrical reactions of degeneration. These latter furnish also valuable information as to prognosis.

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(1) The Lancet, Dec. 15, 1906.

As to the significance of galvanic response, Head and Sherren make the important statement: "(1) If the lesion is complete the response is the full reaction of degeneration, with a characteristic sluggish wave of contraction, altered polar reaction, and *diminished* excitability of the muscle. (2) If incomplete, the wave of contraction is not so sluggish; the polar reactions are usually normal, and there is an *increased* excitability of the muscle. 3. When recovery occurs, voluntary powers and restoration of the faradic irritability occur simultaneously."

Warrington reports several cases of brachial palsy, and deduces the following clinical guides as to location of the division, whether through the roots, trunks or cords of the plexus: (1) Pupillary paralysis results from an intravertebral lesion (within the spinal canal to the vertebral foramen); (2) paralysis of the rhomboidei, serratus magnus, or levator anguli scapulæ indicates a lesion internal to the extravertebral portion, situated in either the intra- or inter-vertebral portion; (3) paralysis of the supra- and infra-spinati muscles indicates a lesion before the formation of the plexus proper.

Warrington's conclusions as to prognosis unmodified by surgical treatment follow: 1. The condition is a serious one, only from 30 to 40 per cent. terminating in useful recovery. That birth palsy of any severity less commonly results in good recovery than the corresponding condition in adults. 2. The pathologic experience shows that the lesion is a severe one caused by laceration, tearing, or tension of nerve tissue, simple pressure being less common. 3. A complete block of conduction with graver prognosis may be diagnosed from (a) a consideration of the extent and kind of anesthesia; (b) a study of the electrical reactions, especially with the constant current; and (c) attention to the extent and distribution of the muscular palsy. The order of gravity of prognosis is lesions of the spinal cord, roots, plexus proper, peripheral nerves. 4. If spontaneous recovery happens, some signs of it are visible within seven months in adult palsy, but in birth palsy signs of recovery may be delayed for a longer period, but are complete within a year."

As to *treatment,* Warrington says that if recovery occurs signs of it are visible within about eight months, but a considerably longer time may be required before treatment is discontinued. In all cases mechanical treatment should be thoroughly tried before operation is advocated. The success of such treatment depends upon the nature and extent of the nerve lesion. In complete nerve division recovery by mechanical treatment is all but impossible. In milder cases, without division of the nerve, "the absorption of callus, the yielding of adhesions and the reduction of a dislocation which presses on the nerve may result in recovery from paralysis. The mechanical treatment consists in keeping the over-stretched muscles completely relaxed, and must be continued without intermission for some months. The object is to antagonize the functioning muscles and to prevent degeneration of muscular tissue which rapidly occurs when it becomes elongated by posture. Where paralysis has lasted several weeks there is little chance of complete recovery unless the elongated muscles are relaxed, and where this procedure is omitted very valuable service has been withheld from the patient. The muscles should be systematically and energetically massaged. Massage applied with the wrist hyper-extended is as useful as it is futile when the muscles are allowed to remain in their elongated state. The constant current appropriately administered is sometimes useful. If the early treatment has been efficient and *no improvement* takes place in seven or eight months an operation may be advised if the clinical signs indicate that the lesion is accessible."

As to *operation,* the results of late years are reassuring. In some cases it has been found necessary merely to free the nerve trunk from inflammatory tissue. Nerves may be united by an end-to-end junction, or nerve anastomosis may be resorted to. In infantile cases anastomosis is the only operation possible. From a survey of the literature one may conclude that almost every method of nerve-junction has been successfully accomplished. Without further data generalizations are impossible, but in operations on the brachial plexus we are scarcely justified in doing more than (*a*) end-to-end junction; (*b*) nerve anastomosis; and (*c*) auto- or homo-implantation. The nerves should be most gently handled and the stitching should be sufficiently accurate to prevent the ingress of fibrous tissue between the axis cylinders. Where, from extensive dissection in fibrous tissue, adhesions may be feared, the nerve-ends can be surrounded by sterile foil. When the operation has been completed and the wound healed massage and the postural shortening of the lengthened muscles should be practiced.

SCIATICA.

Treatment. Archibald G. Hay[1] recommends the injection of normal salt solution into the sheath of the sciatic nerve in cases of sciatica, and reports four cases with cures. The injection is made at a point in the gluteal fold midway between the great trochanter and the ischial tuberosity, into the nerve itself, and 10 c.c. of saline solution is rapidly introduced. The puncture is sealed with collodion. The injection is repeated on alternate days for three to five times.

Brissaud, Sicard and Tanon[2] are decidedly of the opinion that injections of alcohol into the sciatic nerve for sciatica are not to be advised, since motor disturbances of a grave sort are apt to follow. In a second article[3] they confess to having treated two cases of sciatic neuralgia by the method with bad results, the report of Schlösser of Munich of 36 cases with 36 cures by this treatment to the contrary notwithstanding.

Louis Hauck,[4] who learned the method at Schlösser's clinic, on the other hand, advocates the use of alcohol injections in sciatica. "In sciatica the injections are made according to indications, upon the trunk of the peroneal nerve (under the head of the fibula), or of the external saphenous nerve (at the calf), or upon the trunk of the sciatic nerve (at the thigh just under the gluteal fold or in the great sciatic notch). In the case of mixed nerves, such as the sciatic, a 40 or 50 per cent. solution of alcohol seems to suffice to relieve the pain and is less liable to produce permanent paralysis, although with proper care this should not occur even with the stronger solution."

Peripheral peroneal paralysis. James H. Lloyd[5] reports

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(1) Glasgow Med. Journal, May, 1907.
(2) Rev. Neurol., Mar. 30, 1907.
(3) Rev. Neurol., June 30, 1907, p. 633.
(4) St. Louis Med. Review, May 11, 1907.
(5) N. Y. Med. Journal, Dec. 22, 1906.

paralysis of the peroneal (external popliteal) nerve following childbirth. The patient on the ninth day of confinement complained of numbness and tingling in the left leg. This had been present since the birth of the child, but she had not called attention to it. She was found to have foot-drop, and there was anesthesia over the dorsum of the foot and the anteroexternal part of the leg over the peroneal muscles. Paralysis was limited to the muscles supplied by the peroneal nerve, and some pain about the hip. The only objective anesthesia was in the region supplied by the peroneal nerve. For some days the patient continued to complain grievously of pain. Electrical examination showed complete reaction of degeneration.

The sacral plexus is formed from the three upper sacral nerves and part of the fourth, and receives, in addition, from the lumbar plexus, the lumbosacral cord. This lumbosacral cord, of which the peroneal nerve is merely the continuation, in passing from above downward into the pelvis, crosses over the brim at a point where it may readily be compressed under some circumstances. Hence such peroneal paralysis may occur following childbirth.

MULTIPLE NEURITIS.

Julius Grinker[1] reports two cases of multiple neuritis and discusses their *differential diagnosis* from progressive muscular atrophy, which they may simulate. These are cases in which the sensory symptoms are either absent or insignificant, and the muscular atrophy extensive. In progressive muscular atrophy the muscles are affected according to their physiologic grouping in the cord, while in peripheral neuritis they are affected according to their innervation, regardless of segmental grouping. The first case was an alcoholic who developed painless atrophy of muscles in the upper extremities, followed in eight months by similar wasting and paralysis in the lower extremities, but now with sensory disturbances. Within four months, recovery sufficient to permit resumption of work occurred; but two months later there was a relapse, and various cranial nerves became involved. He again entered the

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(1) Jour. Am. Med. Assoc., Mar. 9, 1907.

hospital, and improved in every way except that certain atrophies remained which did not differ essentially from those seen in progressive muscular atrophy. The second patient showed wasting and loss of power in the small muscles of the left hand, later of the right, followed by atrophy in the forearms and shoulders, with no noticeable disturbances of reflexes, coördination or sensation. Yet after death pieces from various cord levels showed no cord changes, while the peripheral nerves (sciatic, median, and ulnar) showed distinct degeneration. A third case reported is one of progressive muscular atrophy resembling multiple neuritis. The writer's conclusions follow: "1. Clinically, multiple neuritis may simulate a spinal atrophy as regards distribution of paraylsis, absence of sensory symptoms and protracted course. 2. Progressive spinal muscular atrophy may resemble multiple neuritis in the presence of pain, remission of symptoms and subacute course. 3. Etiology and course are still our best guides in the clinical diagnosis of the various muscle atrophies."

Surgical Treatment. Basil Kilvington[1] has made an investigation of the surgical treatment of certain paralyses, as those of the bladder. As a result he advocates nerve crossing within the spinal canal. In the case of the anterior root, recovery can follow a lesion anywhere in the neural canal if the ends be brought into satisfactory apposition. Up to the present the author is not aware that nerve crossing has even been suggested within the spinal canal, but it is both possible and practical. He has performed the operation on dogs, and has sutured the central ends of one of the limb nerves to the peripheral ends of the nerve supplying the rectum and bladder, with very encouraging results. Several animals were operated on, but only three which healed by first intention were kept. The after-results produced were some awkwardness in the hind leg on the side operated upon, which partly disappeared in time. Nothing peculiar about the bladder function was noted.

The surgical possibilities are manifest. "The experiments show that the bladder, and presumably the rectum, can be innervated by other of the spinal nerves than those

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(1) Brit. Med. Journal, April 27, 1907.

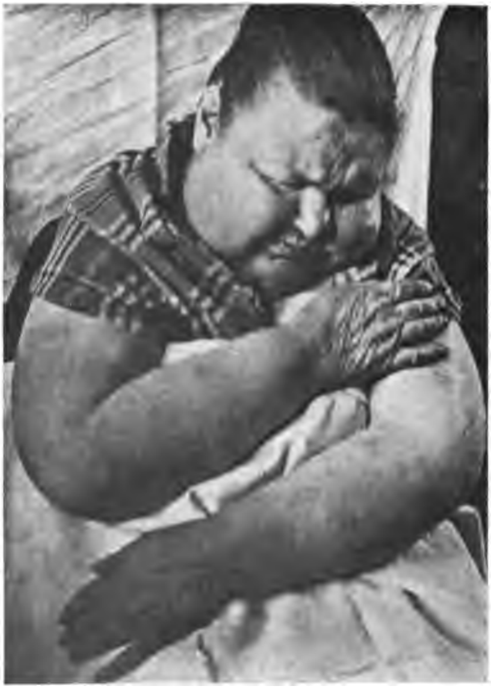

PLATE X.

Adiposis dolorosa in an imbecile, blind and epileptic. (Page 201.) [From the article by A. Prunier, Nouvelle Iconographie de la Salpêtrière, 1907.]

which normally supply them. In fracture-dislocation of the spine the trouble and danger result from the paralysis of the bladder. If it were possible to innervate the pelvic viscera in such cases by nerves above the spinal lesion, bringing them again under voluntary control, many deaths would be prevented, much inconvenience and misery would be relieved. This can be done in some instances by surgical intervention. In man there are two bladder centers, one sending impulses through the lumbar roots, and the other by the second and third, and possibly the fourth, sacral. The sacral nerves are the more important, and should be used in performing nerve anastomosis. It is most important to innervate the pelvic floor in addition, because the bladder muscle itself is not directly under the control of the will. Micturition is a reflex act started by a voluntary contraction of the abdominal and perineal muscles. Summing up our facts, we find (1) it is possible to satisfactorily innervate the pelvic structures by means of a nerve arising higher up from the spinal cord; (2) that the eleventh or twelfth, and possibly even the tenth dorsal nerve, can, in man, be sutured directly to the second, third, and fourth sacral nerves."

Direct end-to-end suture is to be preferred, as nerve bridging adds to both complexity and possibility of failure. The identification of the different nerve-roots is most easily made by applying a very weak faradic current by means of sterilized electrodes, and noting the resulting contraction. The nerves supplying the bladder cause priapism when stimulated, and so may be identified. Nerve crossing should be done as soon after injury as possible, after subsidence of shock and the acute symptoms. Postponement simply gives time for sepsis to arise, and delays the ultimate cure. [This paper was considered under peripheral nerves, since it really deals with them, though the malady to which this surgical method is applicable is traumatic transverse myelitis.—ED.]

BERIBERI.

Etiology. L. H. Fales[1] discusses the etiology and prevention of beriberi, which he defines as an acute or subacute tropical, endemic or epidemic disease, chiefly affecting the ends of the sensory and motor nerves. Interesting from the standpoint of etiology was the epidemic in the Bilibid prison at Manila. Among 2,000 prisoners there were 52 cases in December, 1901, and 167 cases in January, 1902. All defects in sanitation were corrected thoroughly, and the prison was disinfected several times. Floors and walls were scrubbed with 1 per cent. carbolic acid, without result, and all cases were promptly isolated, while those not infected were given baths of 1,000 to 2,000 bichlorid solution. Before eating each prisoner washed his hands in a bichlorid solution. Though this was done religiously for several months, the epidemic continued unabated, and in August, 1902, there were 571 cases, with 24 deaths; in September 522 cases and 31 deaths, and in October 579 cases and 34 deaths. In October another ration was introduced. Beriberi at once began to decline, so that in December, 1902, there were only 89 cases, and during the first 15 days of January, 1903, only 4 cases, with no deaths. The old ration differed from the new in that the bread was doubled, the rice was halved, while more vegetables (potatoes and onions) were given, the increase in the former being about 30 per cent. and in the latter 400 per cent. Fales believes that the reason beriberi attacks those who make rice the main diet is because rice contains very little potash salts. The lack of these salts makes infection by an unknown organism very easy. Those eating freely of vegetables are far less likely to acquire the disease.

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(1) Jour. Am. Med. Assoc., Mar. 2, 1907.

Ira A. Shimer,[1] Captain U. S. Army, writes of beriberi on the Isthmus of Panama. He believes it to be a "place infection" of microbic origin, though he does not pretend to state whether a bacterial toxin made outside of the body is taken into it, or the germ itself gains entrance and produces its toxin *in situ.* The Japanese diet changes prove nothing as to etiology, since together with the changed ration a large number of hygienic reforms were also instituted. As a rule beriberi attacks chiefly persons living in damp, ill-ventilated, and overcrowded buildings.

Shimer then describes the forms occurring in the Isth-

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(1) Jour. Am. Med. Assoc., Mar. 2, 1907.

mus, which, like those of the Orient, are the dry, atrophic or paraplegic type, the wet or dropsical type, and the mixed forms. The symptomatology of each form is succinctly summarized. In all cases the paralyses and atrophy of voluntary muscles play a minor part as to the life of the patient. The real danger depends upon the involvement of the cardiopneumogastric innervation. Patients who are up and around one day, apparently doing well, suddenly collapse and are dead the following day. Therefore the prognosis should be guarded, since apparently mild cases may suddenly develop serious symptoms.

Beriberi was unknown on the Isthmus prior to 1887, when a number of Chinese and African contract laborers were introduced by the French. In July, 1887, there was the first death on the canal, and in December the first one in Panama City. Since 1887 it has been not uncommon. Shimer reports seven cases, some of which showed anchylostomiasis, postmortem. An example of place infection with beriberi is the Boredas prison. The ground floor was poorly ventilated and often overcrowded. Beriberi was common. When, in October, 1905, the prisoners were transferred from this floor into another portion of the same building, beriberi stopped.

Treatment. Victims should be removed from damp, ill-ventilated and overcrowded habitations, to dry, well-ventilated rooms. They should be well fed, but bulky articles of diet should be eliminated. The medical treatment is entirely symptomatic. Digitalis and strophanthus for the heart, with general tonics, massage, and electricity, are indicated.

Justus M. Wheat,[1] writing of some experimental work upon beriberi, comes to the following conclusions:

"1. Beriberi is an acute infectious disease, transmitted by direct infection, probably always through a skin abrasion upon some vulnerable portion of the body.

"2. The specific organism causing the disease is an exceedingly small bacillus, usually seen in pairs, but not encapsulated, and is found in the cerebrospinal fluid and blood only during the acute or inflammatory stage of the

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(1) N. Y. Med. Jour., March 31 and April 6, 1907.

disease, after which time it rapidly disappears from these tissues.

"3. The favorite habitat is virgin soil protected from sunlight. It is a slightly facultative anaërobin, and not very viable, being speedily killed by exposure to sunlight.

"4. The former classification of the disease as *beriberi hydrops* and *beriberi atrophica* is erroneous. There is but one form of beriberi, which is characterized by three stages: 1st. The stage of invasion. 2d. The stage of exudation, beginning in the cord and following by toxic motor and sensory paralysis of varying extent, which in typical cases proceeds to the third, or the stage of compensation, in which the disappearance of the bacilli and the toxic stage results in the rapid (usually) disappearance of the dropsy in cases in which softening and dilatation have not progressed to the degree of permanent loss of compensatory action.

5. The degenerative cord and nerve lesion is long present in all moderately severe cases, resembling the lesion in tabes in this particular, and even though the heart lesion *appears* to have subsided while the patient is leading the inactive life of a convalescent, the damage to function is permanent. This explains the numerous cases of sudden and unaccountable death.

"6. That a toxin or toxalbumin should be isolated is the rational conclusion, and that an antitoxin curative in the period of germ activity and before degenerative change has taken place is the inevitable reward for properly directed effort."

MISCELLANEOUS NERVOUS DISEASES.

ACROPARESTHESIA.

Etiology. W. W. Lesem[1] has studied 200 cases of acroparesthesia from the clinic of Joseph Collins in New York. Of these, 94½ per cent. occurred in women, mostly between 30 and 40 years of age. Less than 16 per cent. developed before 30. Heredity, syphilis, rheumatism, abuse of alcohol, excessive use or wetting of the fingers are all unimportant causal factors. In 33 per cent. a

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(1) Med. Record, Sept. 1, 1906.

history of obstinate constipation was obtained, while the remainder, 20 per cent., complained of chronic dyspepsia. He believes that the vast majority of cases suffer from autointoxication. Faulty metabolism and elimination, and autointoxication may be considered the most important causes. [With this conclusion the Editor is not in accord.] Lesem also believes that vasomotor paresis, arteriosclerosis, or excessive alkalinity of the blood stand in a causal relation to the disease.

Symptoms. These vary from transient tingling in the fingers, hands, arms, toes, feet or legs, to intense burning and pricking sensations in the same localities. Even the chest and hand have been involved, but only after implication of the extremities. In the more severe forms, pain may be so intense as to make sleep impossible, walking difficult, and the use of the hands agonizing. *Physical signs* are absent in the mild forms. In severe cases areas of tenderness are found over the vertebral borders of the scapulæ, beneath the clavicles, and over the superficial nerves of the extremities. The deep nerves are not involved. Diminution of the tactile sense of the hands, fingers or toes is of frequent occurrence. Vasomotor paresis accompanies the majority of all well developed cases. Not infrequently multiple neuritis develops in the severer forms.

The routine *treatment* of acroparesthesia in Joseph Collins's clinic consists "in the administration of 10-grain doses of calomel every fifth night (*sic*), followed by a purge in the morning, or the daily use of cascara as a substitute. Three times a week the patient receives an application of the static spray. The medication in vogue consists of the so-called black mixture:

℞	Sodi. brom	℥ss.
	Pepsin	gr. xv.
	Carb. ligni	ʒiss.
	Olei menthæ viridis	gr. iii
	Glycerin	ʒiii
	Aquæ	ad. ℥ii

Sig. Teaspoonful three times a day.

"In but few instances has this procedure ameliorated the suffering or shortened the disease. By far the most satisfactory results have been obtained by the use of increasing doses of strychnin combined with 15-grain doses of sodium bromid three times a day."

DISORDERS OF SLEEP.

Hugh T. Patrick[1] describes a few disorders of sleep. The first disorder mentioned has been called nocturnal palsy, night numbness, sleep numbness, waking numbness, and *acroparesthesia nocturna.* "The patient wakens in the night with one or more extremity numb, tingling, and more or less paralyzed. This paralysis may be complete, but is of short duration, disappearing after some minutes of rubbing, slapping or shaking. The causes of this disorder, approximately in the order of their importance, are neurasthenia and general nervousness, malnutrition and general debility, the menopause, rheumatism, cold and wet, anemia, arteriosclerosis, hysteria, tobacco, gastrointestinal disorder, gout, Bright's disease, and diabetes. The next disorder is what the author calls *painful awakenings.* The patient wakens suddenly, with a feeling of intense apprehension or panic, as if something dreadful were about to happen or had just happened. There is apt to be palpitation, tachypnœa, generalized tremor or, more frequently, a sensation of trembling without visible tremor, sometimes cold perspiration and a feeling of great prostration. As nearly as may be ascertained, these attacks have no relation to nightmare or bad dreams. They occur principally in the course of functional nervous disease, and, although most distressing, are entirely innocuous, and nearly always disappear with successful treatment of the general nervous condition. However, sometimes heart disease, Bright's disease, arteriosclerosis or adenoids stand in a causal relation to the disorder.

The author then describes different types of what he ventured to call *indormescent shocks.* They occur just as the patient is falling asleep, or immediately after. They may be divided into motor and sensory, although sometimes the two are combined in the same patient or in the same shock. In a mild degree the motor shock is familiar to every one, simply consisting of a sharp muscular start

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(1) Ill. Med. Journal, December, 1906.

or jerk about the time one is falling asleep. The starts in this degree may be called physiological, as nearly every person has experienced them, but they sometimes reach an intensity which makes them distinctly pathological. In such a case the sudden muscular contraction may be so violent and so generalized as literally to throw the patient into the air and turn him over in bed, and in some cases these jerks may be repeated ten or twenty times before the patient finally passes into a sound sleep. The sensory shocks are very apt to be related to the special senses. A typical case was that of a neurasthenic patient who was awakened by what seemed to be the discharge of a pistol inside of his head. He was conscious of a tremendous crash, like an explosion, and, at the same time, apparently saw a vivid flash of light. He sprang from the bed, was terribly frightened, sat up the rest of the night, and for weeks thereafter retired with great timidity. Other similar cases are also recounted. The author has never known the particular disturbance to be a symptom of organic disease. The disorder of sleep always disappeared with the disappearance of the nervousness and a return to general good health.

RAYNAUD'S DISEASE.

F. Sarvonat,[1] writing on the etiology and pathogenesis of Raynaud's disease, finds that age figures but slightly as a causal factor; that sex is important in that the disease predominates in the female; that some occupations, exposing the patient to frequent variations of temperature and the hands to cold water, lead to it; that heredity is a factor; and that it is allied to the nervous circulatory group of diseases. The theories of pathogenesis are the nervous, the vascular, and the toxic. The author believes that in the majority of cases a toxic influence acting upon the hypersensitive vasomotor nerves is at the bottom of the malady.

HUNTINGTON'S CHOREA.

Arthur S. Hamilton[2] discusses Huntington's chorea. Though regarded as rare, he has seen seventeen cases,

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(1) Gazette des Hôpitaux, June 8, 1907.

(2) Jour. of the Minn. State Med. Assoc., and the Northwestern Lancet, Nov. 15, 1906.

twelve being observed in the State Hospital at Independence, Iowa. The disease usually shows itself in patients from 35 to 45 years of age, and is strongly hereditary. The onset is gradual, sometimes the choreiform movements appearing first, sometimes the mental impairment. Though the chorea begins locally, it early becomes generalized. Gait is markedly disturbed, and walking may be impossible. There is no wasting of muscles, disturbances of sensation, or electrical reactions of degeneration. The mental change is a gradually increasing dementia, often associated with great irritability, and even distinct delusions of persecution. In the end-stages the patient is confined to bed, but death usually occurs from some intercurrent disorder. Treatment is of no avail. The pathology is still undetermined, but the condition is probably a cortical or a subcortical sclerosis. Hamilton reports five of his cases fairly completely.

Clarence King[1] prefers the title "hereditary chorea," and calls attention to the fact that the disease was discovered by Dr. C. O. Waters, of Franklin, N. Y., in 1841, or 31 years before Huntington described it. King regards the disease as rare, only 150 to 200 cases having thus far been reported in this country. So decided is King as to the influence of heredity that he would not make a diagnosis without it. From the evidence at hand, rheumatism, pregnancy, hysteria, the emotions, and other functional nervous diseases must be of slight etiologic value. The disease is not a neurosis, though its pathologic anatomy is obscure. Reports of postmortem examinations by Huber, Macleod, Phelps, Rusk, Spiller, Stier and Osler suggest that the disease depends upon a sclerotic or degenerative change, with moderate atrophy of the motor convolutions, secondary to some organic lesion of a variable nature, directly affecting the cortex. The *differential diagnosis* must eliminate Sydenham's chorea, athetosis, the ataxia of paresis, multiple or combined sclerosis, and simple chronic adult or senile chorea. No treatment is known or cure recorded. Arsenic, hyoscin, galvanism and prolonged rest have been tried without success. The bromids merely hasten mental decay.

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(1) Medical Record, Nov. 17, 1906.

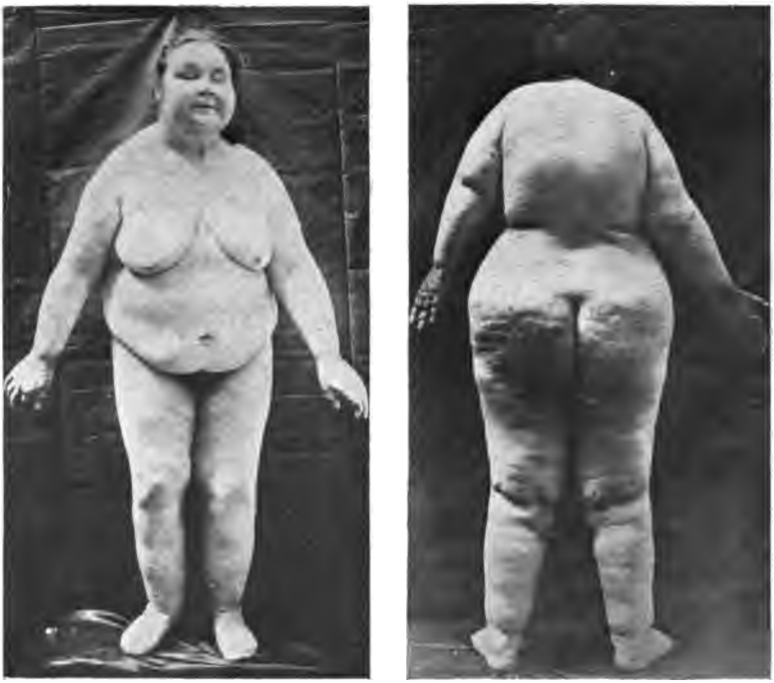

PLATE XI.

Anterior and posterior views of the patient shown in Plate X. (Page 201.)

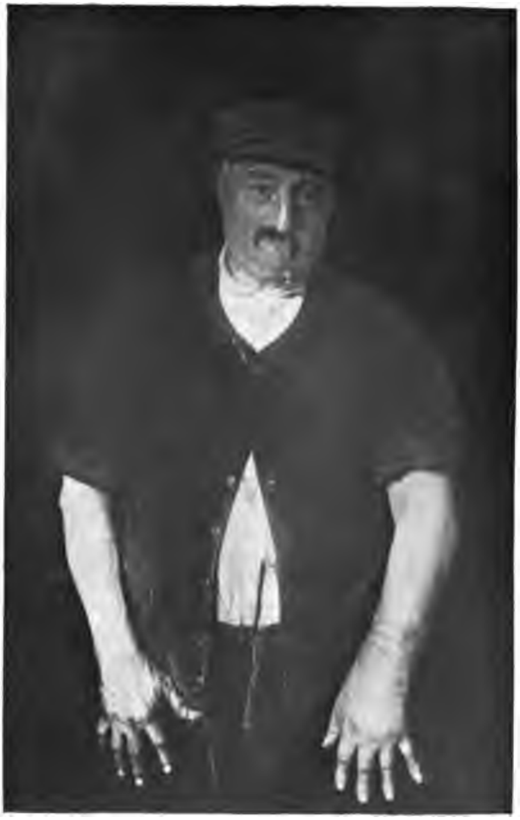

PLATE XII.

Chronic Trophedema of traumatic origin (G. Etienne). (Page 202.)

GIANTISM.

Charles Hudovernig[1] makes a supplementary report concerning a case of precocious giantism showing unusual early development of the sexual organs, thus leading to the diagnosis of disease of the hypophysis cerebri. Treatment for ten months with thyroid extract alone, and for ten succeeding months with thyroid extract and iodin, was without effect; indeed, the boy kept on rapidly growing bigger in every way. Finally, testicular extract not being available, he was put on ovarian tablets for ten months, with excellent results. Both his physical and mental condition improved.

DERCUM'S DISEASE.

L. Haskovec[2] reports a case of Dercum's disease rather amply in a woman, 62 years of age, without hereditary stigmata. The disease, developing at the menopause, was characterized by pain and disturbances of general sensation, crises of weakness, disturbances of the sympathethic system, transitory edemas followed by a diffuse hyperplasia of the subcutaneous fatty tissue, painful spontaneously and on pressure; some disturbances of motion, and an elarged thyroid gland. The case would suggest to any one lipomatosis, with a vasomotor neurosis, hysteria or neurasthenia, Quincke's disease, or even asthenic bulbar paresis. Dercum's disease might be classified among the edemas of nervous origin, yet it seems to be a morbid entity. It is very likely that autointoxications caused by functional lesions of the thyroid gland, the genital glands, and perhaps still others, play an important part.

A case of adiposis dolorosa in an imbecile, blind and epileptic, is reported by A. Prunier.[3] The accompanying Plates X and XI excellently illustrate the degree of adiposity. Everywhere there was great sensitiveness to pressure, especially over the more dense lipomatous spots. Passive movements can be made without pain or joint-symptoms, and the joints are not deformed. The vasomotor disturbances were prominent and nose-bleed was frequent. Treatment by thyroid extract was without results.

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(1) Nouvelle Icon. de la Salpêtrière, July-August, 1906.
(2) Revue Neurologique, Dec. 15, 1906.
(3) Nouvelle Iconographie de la Salpêtrière, March-April, 1907.

TROPHEDEMA.

G. Etienne[1] reports the case of a man, 70 years of age, showing chronic trophedema of the left arm caused by trauma. It began with pain in the palm of his left hand, which was bruised, the pain gradually growing worse. A month later a slight swelling appeared (see Plate XII), without any apparent cause, spreading over his hand and fingers, wrist, forearm, elbow, and lower part of the arm proper, in the course of two years. Here the swelling stopped. The color is whitish. The edema is hard, difficult to pit, and leaves no depression. It is painless, the pain being deep-lying, beneath the edema. Sensibility to touch and pain is intact. The author also reports two other cases. A fourth case of congenital origin is reported by E. Long,[2] a primary report having been made in 1901. The patient was a woman of 30 years of age, and showed congenital hypertrophy of the two arms due to the presence of a hard, incompressible edema, extending from the ends of the fingers to the middle of the arms. The right ear and cheek showed a similar edema. Cancerous ulceration having developed on the back of the right hand, the forearm was amputated. Examination showed a simple increase in the different elements of the connective tissue from the skin to the deeper regions. The muscles do not show a uniform infiltration by adipose tissue. The vessels show no appreciable lesion. Long believes the disturbance to be due to a trophic origin from disturbance in the cord.

A fifth case of chronic trophedema is reported by G. Parhon and S. Florian,[3] involving the left leg. The appended photographs in Plate XIII show the condition better than words can describe it. A discussion of its etiology and pathogenesis is given by the authors.

PROGRESSIVE MUSCULAR ATROPHY.

L. Pierce Clark and C. E. Atwood[4] report a case of progressive muscular atrophy of the Landouzy-Déjerine type in a boy 5 years of age. The family history was

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(1) Nouvelle Iconographie de la Salpêtrière, March-April, 1907.
(2) Nouvelle Iconographie de la Salpêtrière, March-April, 1907.
(3) Nouvelle Iconographie de la Salpêtrière, March-April, 1907.
(4) N. Y. Med. Jour., July 20, 1907.

negative. Very unique is the fact that in infancy the patient was unable to suckle properly, milk being supplied to him from a bottle in the nipple of which a large hole was cut so that it could flow passively into his mouth and throat. He began to walk at the age of 14 months, and made the ordinary movements of all the limbs; but the mother never noticed from the earliest time any movement of the facial muscles except compression of the cheeks and contractions in the right levator alæ nasi. The boy cannot whistle, change his facial expression in crying, smile, or articulate labials and linguals. Careful examination of the literature has disclosed no other congenital cases. This case varies from the classic type as follows: First, the malady is congenital and exhibited itself in the facies and inability to suckle. In this respect the case appears to be unique. Secondly, there is some involvement of the muscles of the eyeball. Thirdly, there is no heredity.

William G. Spiller and J. C. Gittings[1] report a case of progressive muscular atrophy of cervicobulbar type occurring with cervical rib. Similar cases of cervical rib have been noted as occurring in syringomyelia and in multiple sclerosis. Spiller thinks it safe to assume that in the case now reported there was some defect of the central motor system, present from the embryonic period, and that the cervical rib is merely another congenital anomaly. It is well known that where one congenital malformation is present, careful search may reveal others.

MYATONIA CONGENITA.

Congenital hypotonia is reported by Carey Coombs[2] in a girl 10 years of age. This is a form of myopathy apparently never hitherto observed in England, though noted in Germany and America. To it Oppenheim has given the name myatonia congenita. Within half an hour of this patient's birth one hand was noticed to be peculiarly flabby and loose, but no other sign of weakness was apparent until she began to walk. Then it was found that her legs gave way under her. At this period her mother often

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(1) N. Y. Med. Jour., Oct. 6, 1906.
(2) Brit. Med. Jour., June 15, 1907.

found it difficult to put the child's shoes and socks on, because the legs were so weak and flabby. Till she was 7 or 8 her ankles were continually turning over. Her hands were so apparently "jointless" that they were a source of wonder to her schoolmates, and her parents were obliged to forbid her "showing off." In spite of the muscular laxity, no dislocations occurred. Examination of the arms shows universal deficiency of power, accompanied by poor development of the muscles. Unlike the muscular changes of acute and chronic anterior poliomyelitis and of most muscular dystrophies, no particular muscles or groups are selected by the process, except that the distal parts of the limb have suffered more than the proximal. There is also a complete absence of contractures seen in many cases showing muscular atrophy. The most striking feature of the case is the remarkable hypotonicity of the muscles, and resulting from this is an abnormal freedom of passive movements about the various joints. "It is at the finger-joints and the wrists that the greatest mobility is found; each finger can be turned backwards till the tip touches the back of the hand just above the metacarpophalangeal knuckle, and similarly the hand can be extended on the forearm till the knuckles touch the back of the wrist. Flexion at the wrist is equally free. The fingers can be spread out from each other widely; the hand can be rolled up into a cylinder with palm inwards and dorsum outwards. The thumb can be distorted into all sorts of fantastic shapes. The forearm can be hyperpronated."

Coördination is good, and the gait shows no abnormality. Fibrillary tremor was sought for and not found. No coarse tremor is present. Both knee-jerks are brisk. There is no ankle-clonus or Babinski's toe-sign. The electrical reactions are normal. There is no diminution or other perversion of sensation. Evidently the fault is primarily in the muscles themselves.

In nearly all details Coombs' case corresponds to those described by Oppenheim under the name of "congenital hypotonia" or "myatonia congenita." The only other writer on the subject is Spiller, whose record of a case includes the only description of an autopsy hitherto performed. Oppenheim is inclined to give a hopeful prognosis based upon the course of the four or five cases he has seen.

"The congenital nature of the complaint, the absence of the familial factor, the generalized distribution of the muscular change, and the tendency of the patients to get better rather than worse, separate it from acute and chronic anterior poliomyelitis, multiple neuritis, the various myopathies, and the pseudoparalysis accompanying syphilitic, scorbutic and other diseases of the epiphyses." Oppenheim has met with no cases except in infancy. Spiller's patient was also an infant, death being due to tuberculosis. Coombs' patient shows certain differences from Oppenheim's description, as well as resemblances. Neither Oppenheim nor Spiller has seen a case over two years of age. The girl was ten years old. In Oppenheim's cases the limbs were not small, whereas in this case they are very thin. Perhaps this difference is due to the relative excess of subcutaneous fat in infancy. In all cases hitherto recorded the tendon reflexes were absent, but present in this case. But in children under two years the tendon-jerks are difficult to observe. In all other points the similarity is complete.

M. Bernhardt,[1] of Berlin, reported a case of myatonia congenita in March, 1904, and now thoroughly reviews the literature of the subject. In the same journal[2] is a further note by William G. Spiller on the case reported by him in 1905 in the Bulletin of the University of Pennsylvania.

PARALYSIS AGITANS.

Carl D. Camp[3] reports fourteen cases of paralysis agitans, with complete postmortem examination in each, and discusses its pathology. For convenience, the recorded findings may be classified into: (1) Those in the nervous system, the brain, spinal cord and peripheral nerves. (2) Those in the muscles. (3) Those in the ductless glands. Having thoroughly reviewed the recorded pathology, he gives the findings in his own cases. "Many of the muscle fibers were swollen, and in cross-section were round instead of having the normal polygonal shape. There was a marked increase in the number of nuclei within the fibers, many of which were smaller, denser and rounder than the normal. These nuclei were frequently in pairs or in long chains. In several cases the fibers showed atrophic changes, and in these cases, usually of long-standing disease, there was an overgrowth of the connective tissue. In many cases the fibers had a hyaloid appearance and the longitudinal striations were unusually well marked, and in many of the fibers there was a tendency to longitudinal cleavage. Examination of the parathyroid glands from two of the fourteen cases showed pathologic changes present. These changes, when considered together with the experimental evidence quoted and the therapeutic results of Berkley, suggest that the parathyroid glands play an important part in the pathogenesis of paralysis agitans."

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(1) Neurol. Centralblatt, Jan. 2, 1907.
(2) June 1, 1907, p. 508.
(3) Jour. Am. Med. Assoc., April 13, 1907.

The author's conclusions follow:

1. Paralysis agitans is not a neurosis, nor is it senility. 2. The anatomic basis of the symptoms, muscular rigidity, tremor, and the symptoms dependent on them, lies in the affection of the muscles. 3. The disease itself is probably a general toxemia, and there is suggestive evidence that it is due to alteration in the secretion of the parathyroid glands.

Joseph Sailer[1] reports a case of unilateral paralysis agitans occurring after hemiplegia. Seven years ago a woman developed right hemiplegia, from which she made a partial recovery. Four years later she first noticed tremor in the arm and hand of the *left* (opposite) side, which has continued to the present time. There is now the typical symptom-complex of paralysis agitans in the left arm, with paresis of the left arm, and tremors in both feet.

A. A. Eshner[2] reports a case of paralysis agitans without tremor, making the third of this sort which he has recorded. The countenance of the patient was fixed in appearance, the shoulders were stooped forward, and there was a tendency to propulsion. Though no tremor was present when the patient was first examined, it subsequently developed. Eshner believes that the permanent or temporary absence of tremor is due to restraint through muscular rigidity.

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(1) Jour. of Nerv. and Ment. Dis., July, 1907.
(2) Medicine, December, 1906.

AMAUROTIC FAMILY IDIOCY.

F. J. Poynton, J. H. Parsons, and Gordon Holmes[1] made a noteworthy contribution to amaurotic family idiocy in *Brain.* They report three cases, two of them with postmortem examination, very elaborately worked out, and present a thorough discussion of the literature. The pathologic conditions in their two cases were substantially the same as those recorded in the more complete of the previously published postmortems. The cell changes were very similar to those described by Risien Russell and others, and must be regarded as typical of the disease. It is undoubtedly a morbid entity, since it has a distinct morbid anatomy. There is strong evidence that it is a primary disease of the nervous elements. These are affected not only throughout the whole nervous system, but also in the dorsal root ganglia and in the retina, and so universally that not a single normal cell can be found. The changes discovered proved that the disease is not a primary affection of the neuroglia; proliferation of the latter merely follows degeneration and destruction of the nervous elements. Moreover, there is no evidence that the changes found resulted from any vascular condition, since there is no visible disease of the vessels, and no signs of either older or recent inflammation.

Relatively the nerve-cells are probably more affected than the fibers. In many systems, as in the direct cerebellar tracts, the dorsal and ventral spinal roots, and the optic system, there may be no visible change in the fibers, though the cells from which they spring are severely altered. From this fact it may be concluded that the affection is a primary cell disease and that alterations visible in nerve fibers are only secondary.

The condition of the nerve-cells, as demonstrated by the elective stains (Nissl's, Bielschowsky's), shows that the interfibrillar protoplasm is very much more severely affected than are the neurofibrils, and suggests that the primary change is a disease of the interfibrillar protoplasm, and that to this the alterations in the neurofibrils are secondary.

As to *etiology,* the disease is not due to arrested develop-

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(1) Brain, Part 114, 1906, p. 180.

ment. If it were, clinical symptoms of the disease would be evident from birth, which has never been observed; and there is no reason why mere arrest of development should produce a progressive and invariably fatal disease. The negative results of bacteriologic examination, and the entire absence of bacterial reactions in the vascular and lymphatic systems suggest that the disease is not due to bacterial toxins. Its family nature and occurrence in the Jewish race only argue against a bacterial origin. The final conclusion, which is supported by the microscope, is that the disease is due to some inherent biochemical property of cell-protoplasm, as a result of which the neurone undergoes degenerative changes which spread consecutively to the nerve-processes.

Warren Tay in 1881 reported in a child a year old, which could hardly hold up its head or move its limbs, and which was mentally defective, a peculiar condition of the macula lutea. He found a peculiar symmetrical change in both maculæ luteæ, namely, a diffuse white patch in the center of which was a brownish-red point. B. Sachs, in 1887, in the *Journal of Nervous and Mental Diseases*, made an independent report of the very same condition, to which he gave the general name of congenital amaurotic idiocy. L. Huismans[1] reports a case of this malady in a three-year-old child. The psychic defect postulated by Sachs seems uniformly present, and appeared in this case. A discussion of the symptomatology and pathogenesis follows, together with a short bibliography.

Cases allied to amaurotic family idiocy are said by Alfred Gordon[2] to exist, two of which he reports. He also offers some suggestions as to the pathogenesis of the affection. The typical clinical picture of amaurotic family idiocy is not always present in its entirety. "The symptoms which may be absent are: Spasticity or increased reflexes or convulsions. Eye symptoms, the family character, the mental deficiency, and finally the early death, are features indispensable to constitute the type of amaurotic family idiocy. On the other hand, there are cases which, although they do not present all the important symptoms of that affection, show nevertheless the majority

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(1) Deutsche med. Woch., Oct. 25, 1906.
(2) N. Y. Med. Jour., Feb. 16, 1907.

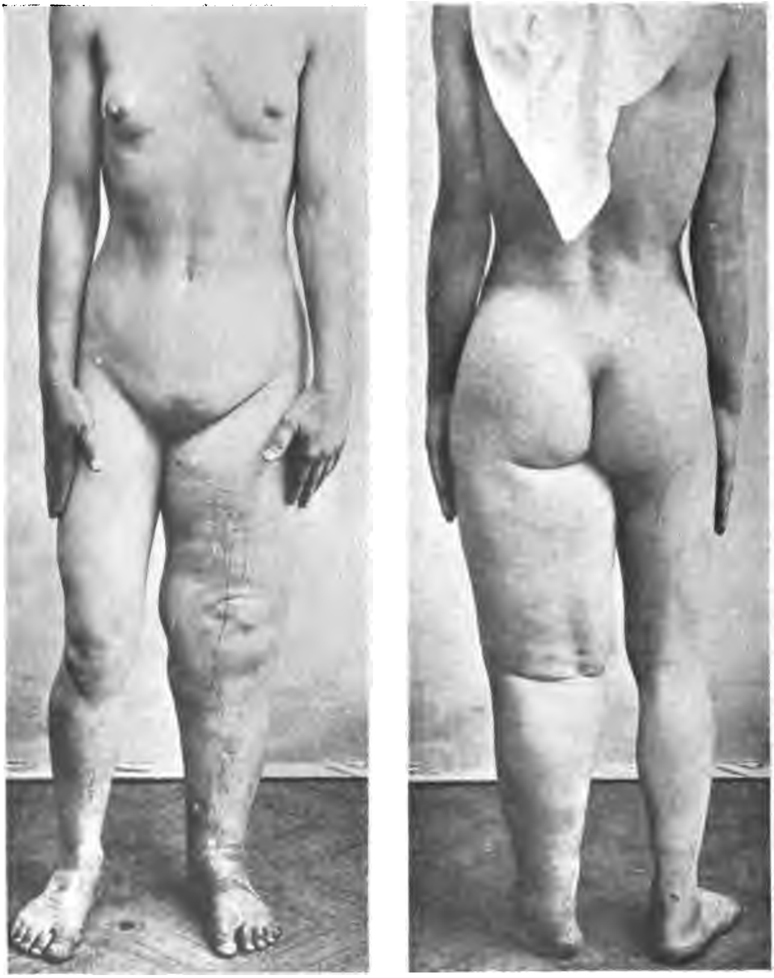

PLATE XIII.

A case of Chronic Trophedema (C. Parhon and St. Florian). (Page 202.)

of them, and must therefore be considered as allied to that disease.

Both Spielmeyer[1] and Spiller[2] report such aberrant cases, and in Spiller's case the cellular changes characteristic of the disease were found postmortem. This patient lived eight years.

The two cases reported by Gordon "are allied to the Tay-Sachs disease first, because the blindness with which they are afflicted was noticed very early in life, and secondly because the mental deficiency was noticed since infancy. While the changes of the eyegrounds are not entirely typical of the amaurotic family idiocy by reason of absence of the cherry red spot in the macula, nevertheless optic atrophy, which is present here, was stated in the majority, if not in all cases of that disease." Clinically Gordon's two cases, a brother and sister of Russian Hebrew parentage, belong to the same category as Spielmeyer's cases, and they can be considered as forms of the same great group of affections called in a general term amaurotic family idiocy.

The stigmata of degeneracy in these two cases are far more extensive than in the Tay-Sachs or the Spielmeyer forms. The boy, for example, presents among other stigmata a very unusual anomaly, viz., polydactylism of all four extremities. A photograph accompanying the article shows this polydactylism and the facial expression of the child. See Plate XIV.

Various views have been advanced as to pathogenesis. Gordon suggests that "it is possible that some profound anomalies in the structure and function of the *ductless glands* are the real cause of the diffuse anomalous condition of the cells of the nervous system." Only two autopsies are reported in which mention is made of such glands, in one of which the thymus was small, and in the other of which the suprarenals were pale, yellow, firm, and had a small cavity in the center. Gordon's two patients showed distinct changes in the thyroid gland, one being enlarged, and the other not palpable at all.

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(1) Neurologisches Centralblatt, 1906.
(2) American Journal of the Medical Sciences, 1905.

MENTAL DISEASES.

GENERAL CONSIDERATIONS.

Increase of Insanity. G. H. Savage, in the Lumleian Lectures, discusses fully the subject of increase in insanity.[1] The author's conviction is that there is a steady increase of the insane, which is definitely related to the progress of civilization. This implies neither hopeless degeneration of the race, nor dread as to the minds of posterity. Experience seems to teach that many of the increasing numbers are attributable to social misfits rather than to material brain disease. Insanity is not a definite entity, and there is no one comprehensive definition of it. What is reasonable conduct in one man under certain conditions may be plain madness in another differently situated. Very many chronic insane owe their position largely to their surroundings, and the part which these have played in the determination of that position must be considered before any reliable statistics can be gathered as to increase or decrease of insanity.

(1) As to insanity dependent upon actual brain disease, it is certain that, whereas in a former generation a great group of the senile insane from gross brain disease was kept at home, this same group is now largely committed to public asylums. The insanity of old age thus represents a large part of such increase. (2) Insanity due to brain intoxication, best illustrated by paretic dementia (general paralysis) without doubt shows a considerable increase. (3) The insanity of the truly neuropathic provides the largest number of long-lived residents in asylums. These patients so afflicted usually come of insane stock, and transmit their neurosis. These patients, even if they recover, show a marked liability to relapse. Such persons are slowly increasing in number. (4) The in-

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(1) Lancet, March 30, April 6 and 13, 1907; Brit. Med Jour., March 16, 23 and 30, 1907.

sanity of habit or association includes a large number of persons who drift into delusional insanity. An idea grows, finally dominates, and practically renders insane its victim. (5) Finally as to what may be called the ordinary forms of mental disorder, mania seems to be on the increase; melancholia seems to show both an increase in the number of patients involved and an increase in the number of melancholic symptoms; dementia is increasing, particularly in the aged, and possibly in the young.

The *causes* of insanity bear upon the matter of increase. The three principal causes are hereditary predisposition, alcoholic excess, and influenzal poisoning. As to heredity, it should be understood that there is no definite form of insanity which is likely to be transmitted, yet it is undesirable that neurotics should intermarry. No statistics are available to show that any increase of insanity depends upon any such cause, though Savage is inclined from his experience to believe that such is the case. Undoubtedly a certain number of idiots are the issue from insane or alcoholic parents, and it may also be said that certain forms of delusional and moral insanity, and the insanity of obsessions almost invariably show inheritance as a partial cause. All such forms are preventable and should not show an increase. As to the question of inheritance in relationship to other forms of insanity, it may be affirmed that melancholia shows a greater tendency to transmission than mania. Of all the forms of hereditary insanity, those associated with hallucinations of the senses and definite obsessions and marked delusions are most common. Consanguinity alone is not to be regarded as very dangerous.

The next generally accepted cause of insanity is alcoholic intemperance, and there is no doubt that excesses lead to direct alteration in the nutrition of the brain and nervous system. Moreover alcoholic parents tend to have degenerate children, idiocy, imbecility, and moral insanity often presenting.

Influenza has been a powerful starting point for the neuroses, and Savage believes it to have been a potent cause in increasing the number of insane. Influenza, like other infective fevers, may set up any form of psychopathy; and there is no specific form of insanity which can be called influenzal. Moreover, in patients who have had previous attacks of insanity, influenza may start a fresh attack.

The following statistics bearing on increase of insanity may be advantageously quoted: "In January, 1906, there were 121,976 certified patients in England, this being an increase of 2,150 over the year 1905. This rate of increase is less than that of the average increase for the previous five years, which was an average yearly increase of 2,807, the average increase for the ten previous years being 2,554. If we compare the returns of the certified insane in 1859 with those of 1906, the numbers are startling. In the former year in England and Wales there were 36,762. Now there are 121,976, that is an increase of 231 per cent., while the population increased during that period only 75.4 per cent. The ratio of certified patients to the general population in 1906 is 1 to 283; that is, 35.31 to every 10,000 inhabitants, whereas in 1859 it was only 18.64 per 10,000. The Scotch Commissioners this year give an account of the increase since 1858, in which year there were 5,824 certified patients, whereas on January 1, 1906, there were 17,450, showing an increase in nearly half a century of 11,606. This increase is confined to the general public asylums, there being a decrease of 624 in the residents in private asylums. In Scotland during the last three years there has been a decrease of insane persons in proportion to the population, for the first time since 1858. Among the causes of total increase of admissions are a large number of senile cases from infirmaries. In Scotland, as elsewhere, the struggle for existence render it more difficult for the poor and healthy to maintain the feeble or insane in their own houses, the residence of such people interfering with the wage-earning capacity of the family. It is noteworthy that—whether there is an increase in other forms of mental disorder or not being doubtful—there is in Scotland, as well as in England, a marked increase in the number of general paralytics. Not a quarter of a century ago the numbers were quite small; now they are large and increasing."

General Prognosis. The prognosis of insanity in general is taken up by A. R. Urquhart.[1] In no disease is prog-

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(1) Lancet, March 2, 9 and 16, 1907.

nosis more difficult or doubtful. Unfortunately, old asylum records afford no data of value. Urquhart's deductions are based on 419 male and 390 female patients, being a total of 809 persons, admitted to the Perth Asylum from 1880 to 1904 inclusive. The percentage of those with hereditary history was 45, while the inclusion of the whole neuropathic heredity increases that number to 72. Yet in a matter of prognosis it must be remembered that heredity works in two directions—for better or worse. Alcoholism had a part in the etiology of a large number of cases also hereditarily tainted, and in them the prognosis is generally unfavorable. General paralysis furnished 40 of the 809 cases; and, of course, in this disease the prognosis is absolutely bad.

Age of onset has a bearing on the outlook. The percentage of recoveries from melancholia tends to diminish as the age-periods increase, while the reverse is true of mania. The slight mental reduction of melancholia in youth is eminently curable, but the deeper reduction of mania is less likely to end favorably. Occupation and former environment have no effect upon the prognosis. As to life expectancy, insanity shortens it. The average age on admission for men was 36 years, and the age at death 54.65 years, as against 63.96 for healthy males; while the average age on admission for women is 38 years, and the age at death is 60.93, as against 66.83 for healthy females.

The general nutrition of the patient furnishes the most certain index of his progress. In asylum life the periodic and exact weighing of patients is a routine practice generally adopted, for it serves as the earliest warning of complications such as tuberculosis. A restoration of lost body weight is one of the first signs of returning health. As to the muscular system, all patients displaying rhythmic, monotonous movements are to be regarded as most seriously damaged. In them the prognosis is bad.

Prophylaxis. The importance of the preinsane stage of acute mental disease is insisted upon by Campbell Meyers.[1] A patient usually requires weeks or months before reaching the boundary line of insanity. The alienist

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(1) Brit. Med. Jour., Oct. 20, 1906.

is not called until insanity exists, and the general practitioner fails to grasp the situation until it is too late to adopt preventive measures. Yet the early symptoms are recognizable. Chief among them are diminished power for sustained intellectual effort and concentration of attention, thus leading to a state of habitual distraction. Ideas do not present themselves as readily or in as rapid succession as in the normal state. The will power and energy are lessened. Morbid sensitiveness supervenes, and emotions are easily thrown out of equilibrium. Mental depression is often marked. Some lose a sense of the proprieties. Natural affections become dulled. Frequent attacks of senseless fear are usual. Insomnia is an early sign, often accompanied by the most distressing dreams when belated sleep finally comes.

Such cases need preventive treatment, and provision should be made for their temporary care in general hospitals. Meyers, by his efforts in Canada, has succeeded in having established in the Toronto General Hospital three rooms with twelve beds for such cases. It is expected that favorable results will follow, just as they have followed similar endeavors in England.

Edwin Goodall[1] makes the same plea for hospital treatment of curable cases of mental disorder, advocating the establishment of mental wards and out-patient departments in the leading London hospitals. He predicts that a large proportion of those who might be received would recover, and, having never been formally committed to an asylum, would thus escape a stigma otherwise their fate. He thinks it likely that not very many of the temporarily detained would ultimately have to be committed to asylums.

F. S. Toogood[2] also advocates detention of alleged lunatics for about three weeks in an infirmary before they are committed to an asylum, since many cases quickly recover their reason. To the Lewisham Infirmary in England, during the ten years from 1896 to 1905, 2,033 cases of alleged lunacy were admitted; of these, 1,020 were discharged cured or sufficiently recovered for their friends to look after them, 804 were sent to asylums, and 209 died. *Alcohol* is directly responsible for a large number of

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(1) Brit. Med. Jour., Oct. 27, 1906.
(2) Lancet, Sept. 15, 1906.

cases—471 patients, or 23.16 per cent. or the total. Of these, 403, or 85.5 per cent., recovered; 18, or 3.8 per cent., were sent to asylums; and 50, or 10.6 per cent., died. Thus alcohol furnishes 39.5 per cent. of the total recoveries; 2.24 per cent. of the cases sent to asylums; and 23.9 per cent. of the total number of deaths.

Delusional Insanity. The dividing line between this group and melancholia is indefinite, many delusional patients being depressed, and many melancholics having delusions. Included in this class are those in whom the delusion is primary, and the depressions, if any, secondary. "Out of 407 admissions, 283, or 69.5 per cent., were sent to asylums; 118, or 29 per cent., recovered; and 6 died. Considering the cases as a whole, it seems strange that 29 per cent. of cases admitted for so grave a condition as delusional insanity should recover in the short period of, say, 17 days. I have no doubt that in some of these recoveries the exciting cause of the breakdown has been alcohol, although there has been no suggestion of tremor or other indications of delirium tremens.

"*Melancholia.* This condition is responsible for 233 admissions, of whom 141, or 60.5 per cent., were sent to asylums; 91, or 39.5 per cent., recovered; and 1 died (a female committed suicide).

"*Mania.* Of 149 cases of mania, 49, or 32.9 per cent., were those of men, while 100, or 67.1 per cent., were those of women; 123, or 82.5 per cent. of these cases, were sent to asylums; 21, or 14.1 per cent., recovered; and 5 cases, or 3.3 per cent., died.

"*Stuporose Conditions.* These conditions account for 141 cases, of which 61, or 43.26 per cent., were those of men, and 80, or 56.7 per cent., those of women; 56, or 39.7 per cent., of the total were sent to asylums; 82, or 58. 1 per cent., recovered; and 3, or 2.1 per cent., died, one of these being a female case of myxedema. This class includes all those cases characterized by loss of memory and by an inability adequately to appreciate environment, and which range from a slight mental confusion to a blotting out of all previous experience.

"*General Paralysis of the Insane.* This class was represented by 83 cases, 79, or 95.2 per cent., being men, and 4, or 4.8 per cent., being women; 72, or 86.7 per cent., were transferred to asylums; 5, or 6 per cent., recovered their mental equilibrium sufficiently to be handed over to their friends; and 6, or 7.2 per cent., died.

"*Senile Dementia.* This condition furnishes 190 cases, of whom 119, or 62.6 per cent., were males, and 71, or 37.4 per cent., were females; 29, or 14.7 per cent., of these were sent to asylums; 68, or 35.8 per cent., recovered sufficiently to warrant their discharge; and 93, or 49 per cent., died.

"*Puerperal Mania.* This condition was responsible for 25 cases. Twenty-one patients were sent to asylums, three recovered, and one died."

Toogood has collected, in his capacity as honorary secretary of the lunacy committee of the Metropolitan Workhouse and Infirmary Medical Officers' Conference, the figures for London of insanity in the last four years. They are as follows:

Years.	Cases.	Sent to Asylums.	Cured.
1902	8,076	3,920	3,560
1903	8,004	3,792	3,443
1904	7,654	3,716	3,155
1905	7,322	3,583	2,877

"These figures show that so far from lunacy having increased in London, it has in four years steadily decreased, being 754 less in 1905 than in 1902, the numbers sent to the asylum from the workhouses and infirmaries being also 337 less."

Toogood insists that no case should be sent directly to an asylum, but that all cases should go through a sifting process at an infirmary half-way house. For cases not paupers, the difficulty arises that such an institution would be immediately dubbed an asylum by the public. If, however, each general hospital had a wing given over to such cases, it would not bring any stigma upon a patient detained within it for two or three weeks.

The Munich Treatment. G. H. Kirby[1] describes the psychiatric clinic at Munich, where the idea and suggestions of Professor Kraepelin are being carried out. The

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(1) Med. Rec., Dec. 22, 1906.

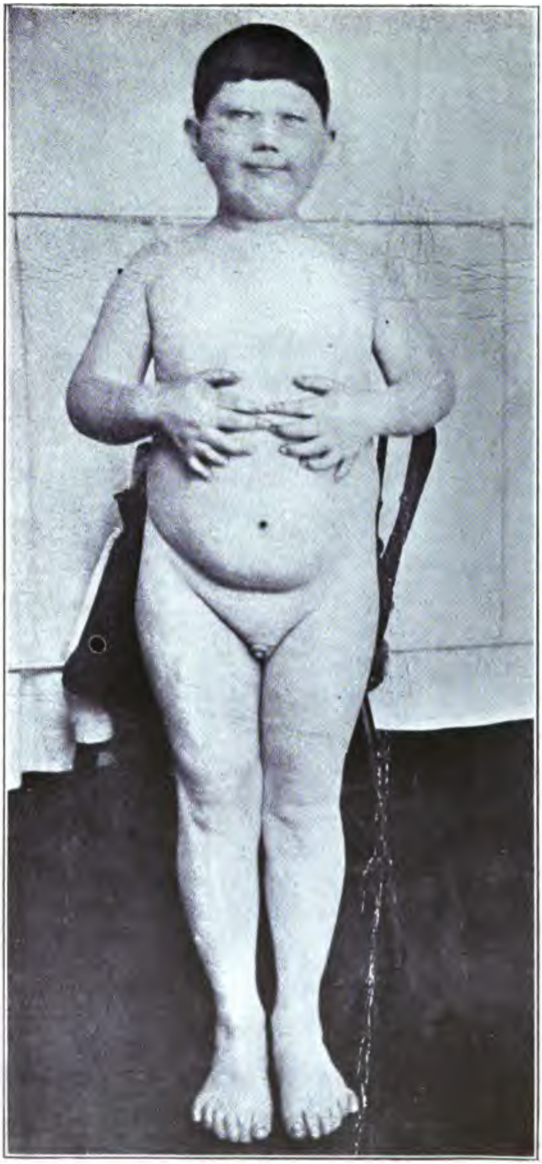

PLATE XIV.

A case allied to amaurotic family idiocy, showing polydactylism. (Page 209.)

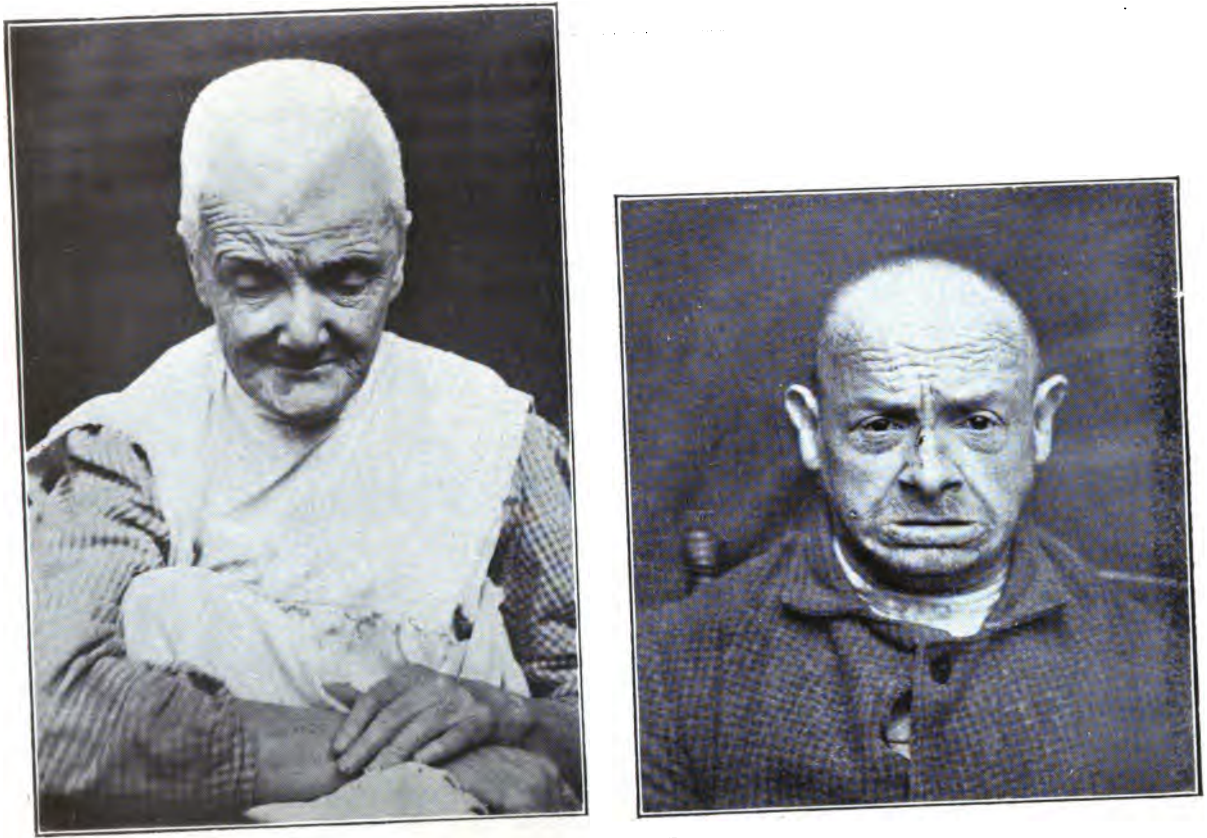

PLATE XV.

A. Emily L., aged 60 years.

B. Tony W., aged 42 years. (Page 223.)

clinical building was designed to accommodate one hundred patients. It consists of a central part, containing the administrative offices and laboratories, and two wings containing the wards. There are only two single rooms for private patients in the whole building, and these are never used for purposes of isolation. The wards contain from ten to twelve beds each, and upon entering them the visitor is struck by the prevailing quietness and good conduct of the patients. Professor Kraepelin ascribes the good order to the method of treatment in vogue, especially to rest in bed, the continuous baths, and also to the fact that most of the patients enter the hospital without going through the hands of the police and the trying circumstances of arrest. Many come voluntarily, and more are brought by friends.

Drugs are rarely prescribed, and then only to meet definite physical conditions; on the other hand, hydrotherapy is carried out in the clinic probably more extensively than in any other institution in the world. Tubs suitable for the continuous bath have been liberally provided, there being no less than eighteen. There appears to be no contraindication to the continuous bath; all forms of excitement and agitations are favorably influenced—manic attacks, delirious alcoholics, anxiety psychoses, and excited general paralytics are treated by this method. No drug sedatives or restraint of any kind are used.

The psychological laboratory organized by Kraepelin has been productive of results. "As a measure of the amount of mental work which an individual is capable of doing, Kraepelin and his pupils have developed and extensively applied the so-called method of continuous addition. Sheets with single rows of figures are placed before the patient, who adds each two figures together, and the number of additions accomplished in every minute is indicated by a stroke of the pencil which the patient makes as each minute is called off by the physician. The experiment lasts ten days, the patient works ten minutes daily, adding as many figures together as possible in that time. On alternate days a pause of five minutes follows the first five minutes of work, then after this rest another five minutes' work is done. . . . Curves can be easily plotted to show the results." In a number of cases of traumatic neurosis, in which the patients complained strongly of fatigue, increased fatigibility was actually found present.

"For the study of apprehension and retention, a fairly simple apparatus is used. By means of a shutter arrangement, a number of letters, usually six, are exposed to the eye of the patient for a known length of time. In this way is determined the number of letters correctly perceived and the length of time they are retained in consciousness. By this method it is possible to study the delay in apprehension and the memory disorder of the alcoholics, especially the Korsakoff delirium."

Occupation Therapy. Occupation in the treatment of the insane is advocated by Thomas J. Mober.[1] Patients should not necessarily be assigned to labor similar in character to that which they have been accustomed to perform. It is better to send them out into the open air, and keep them in motion at simple gardening. This develops the power of observation. When, after working a few minutes, the abnormal ideas return, and the patient stops, he should not be coerced, for he may merely become obstinate. Ultimately, if left to himself, he will automatically resume work again. The percentage of patients not likely to derive benefit from occupation is very small, especially if the physically unfit and the extremely demented are omitted.

The author's conclusions follow: 1. Only a small proportion of our patients should be exempt from labor. 2. A careful study should be made of the temperament of each patient before deciding the kind of labor suitable in each case, and the previous occupation or social condition should not be the only determining factor. 3. The carrying on of this work successfully depends in a large measure on the attendants in charge. Attendants should be selected with care and carefully instructed. 4. Members of the medical staff should visit the working parties regularly for the purpose of studying individual cases. 5. Patients should never receive wages. It is possible that such a system may appear to work successfully in some cases, but in the end it is sure to create trouble. 6. In some cases

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(1) Jour. Am. Med. Assoc., May 18, 1907.

it is advisable to encourage patients to learn a trade, so that they may the more easily earn their living after their discharge. 7. Agricultural pursuits and caring for grounds appear to be ideal labor for the great majority of patients. 8. Female patients may with advantage be employed in the vegetable and fruit garden, weeding, picking berries, etc., in addition to the work performed in the day rooms, dormitories and dining rooms. They should also perform fancy work, do all the mending, and assist in the industrial department in the making of mats, mattresses, etc.

GENERAL PARALYSIS, ETC.

Occurence in old Age. M. Olivier[1] gives proofs that general paralysis may involve old men, and he furnishes statistics from the asylum at Blois. Of 139 cases, three were between 60 and 70 years of age. The diagnosis is difficult, cases being usually regarded as due to senile dementia from cerebral arteriosclerosis.

Classification. P. Sérieux and M. Ducoste[2] present a statistical study of the different clinical forms of general paralysis. These they divide into: 1, simple, demential (without delirious disturbances); 2, maniacal; 3, depressive; 4, expansive and megalomaniac; 5, hypochondriacal; 6, circular; 7, with ideas of persecution accompanied by hallucinations; 8, sensorial. The forms may be pure or combined in any proportion. Their statistics are based on 150 male patients, and the three largest groups are, the demential (24 per cent.), the expansive and megalomaniac (27 per cent.), and the sensorial (24 per cent.). Each of these forms is described symptomatically.

Symptomatology (a) *Blood-pressure.* G. L. Walton[3] has studied the blood-pressure in general paralysis of the insane, chiefly with the Riva-Rocci instrument and with a 9 cm. armband, the 5 cm. band being unreliable. There is a general belief that the blood-pressure in paresis is low, but Walton's studies tend to prove the contrary. His conclusions follow:

1. The average blood-pressure in paresis, taken as a

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(1) Rev. de Psych., Aug., 1906, p. 300.
(2) Le Prog. Med., Mar. 16, 1907.
(3) Jour. Am. Med. Assoc., Oct. 27, 1906.

whole, is high. 2. This is doubtless due to the prevalence of atheroma, with its cardiac and renal accompaniments. 3. The average blood-pressure in cases of paresis without atheroma, cardiac enlargement or renal disorder, is probably somewhat lower than that of health, but the variations are so great that it cannot be said to be uniformly low. 4. The test is not likely to prove of great practical value in the differentiation of paresis from other nervous disorders, though here, as elsewhere, it is of great value in estimating the circulatory condition of the individual. 5. These observations are too few to establish a rule with regard to the blood-pressure in varying emotional states. As far as they go, however, they tend to show (a) that the excited states of paresis are as likely to be accompanied by high as by low pressure; (b) that mental depression is accompanied by high oftener than low pressure, but that it is not incompatible with low pressure; (c) while the average pressure in euphoria is perhaps somewhat lower than in the other mental states of the general paralytic, it is not inconsistent with high pressure, or with pronounced atheroma with its cardiac and renal accompaniments.

(b) *Pupillary Reactions.* G. H. Kirby[1] describes some special work now being done at the psychological laboratory at Munich in Kraepelin's clinic. "The pupils are viewed through a small telescope, definite amounts of light can be thrown in either eye, and the alteration in size of the pupils accurately measured in millimeters. The behavior of the pupil can also be easily observed under emotional stimuli and painful cutaneous impressions." The reaction to which special attention is here called is designated as the *secondary light reaction,* and is described as follows:

"Illumination of, say the right pupil, with a definite amount of light, causes, as would be expected, a contraction of the left pupil as well as the right (ordinary consensual reaction). If now the left pupil is illuminated with the same strength of light as the right, then the left pupil contracts still further, and likewise the right pupil, and in a sufficient degree to be measured. In other words, a definite amount of light thrown into one eye

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(1) Med. Rec., Dec. 22, 1906.

causes both pupils to contract equally, and then a further equal contraction of both pupils occurs when the other pupil is subjected to the same amount of light. This constitutes the secondary light reaction. The failure of this reaction was not observed in any healthy individual nor in any of the psychoses examined, except general paralyses. A series of general paralytics was examined in whom the ordinary light reaction was still present.

"In 96 per cent. of these cases the secondary light reaction was absent. The investigator thinks that this sign is of importance because, in a series of over 500 general paralytics examined in the ordinary way, 18 per cent. of the pupils reacted normally, and in another 40 per cent. a reaction to light was still present."

Treatment. Since Koch's discovery of the value of atoxyl in the treatment of sleeping sickness, it has been used for other ailments in an empirical way. Thus V. Babes and A. Vasilin[1] have used it with advantage in pellagra, and Spielmeyer has used it in general paralysis of the insane for four years. Lassar[2] speaks of it highly in the treatment of syphilis, and Uhlenhut, Hoffmann, and Roscher[3] have also written concerning its therapeutic value.

Spielmeyer began using it systematically in 1903 and 1904 in general paralytics. The indications for its use seem to be general physical weakness and purulent skin affections. He prescribed it especially for patients much run down physically, who were in a restless and excited mental state. The great restlessness of some of the cases of general paralysis, their lack of cleanliness as to bodily functions, favor furunculosis and carbuncles. To such patients were given 0.2 grams (3 grains) of atoxyl, and the skin rapidly healed, and not infrequently the general nutrition improved. Spielmeyer has thus treated 10 cases of general paralysis of the insane, from 4 to 6 to 12 weeks at a stretch. The initial dose of 0.1 to 0.2 grams (1½ to 3 grains) has been in several cases gradually increased to 0.36 grams (5½ grains); and this dose was

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(1) Berl. klin. Woch., July 15, 1907.
(2) Berl. klin. Woch., June 3, 1907.
(3) Deutsch. med. Woch., June 3, 1907.

given to two patients for three weeks, once daily. No effect, however, was exerted.

Spielmeyer calls attention to the resemblances between the *Spirochaeta pallida* and the trypanosomes which atoxyl so markedly affects, and also his investigation of "trypanosome tabes" in dogs.[1] But the suggestion from these studies, that atoxyl might influence metasyphilitic disease, has, on experimentation, led to nothing.

Juvenile General Paralysis. C. H. Fennell[2] believes that juvenile general paralysis is more common than is supposed. He has analyzed over twenty cases observed by himself, in eleven of which postmortem examinations were made. He places the percentage of general paralytics in any series of imbecile children at about 5. In the records of 18 months at a large institution for imbeciles, he has found the percentage at postmortem to be as high as 17. In all cases the disease is the outcome of stress operating upon a nervous system tainted with hereditary syphilis, and he has never met with a case from which it could be excluded. The age of onset of the malady, given as 17 years by Mott, is regarded as too high. One patient is instanced in which symptoms appeared at 8½ years, and another child is referred to in whom signs were well defined at 7½, a subsequent postmortem observation confirming the diagnosis.

The cardinal physical signs are iridoplegia, affection of speech, alteration of deep reflexes, and muscular tremors. Of these the first is generally observable early, and is frequently suggested by inequality and irregularity of the pupils. A curious facial expression of preoccupation and worry is typical of the earlier stages of the disease, contrasting very distinctly with the cheerful unconcern or blank indifference of the ordinary imbecile. Definite delusions are less common than in adults, and megalomania is rare. "Periods of acute excitement or depression are occasionally met with, but in the majority of instances the mental record is one of dementia of indefinite onset, unbroken by maniacal or melancholic accesses. As the disease makes headway, diagnosis increases in difficulty. Physical weakness becomes more marked, until the sufferer

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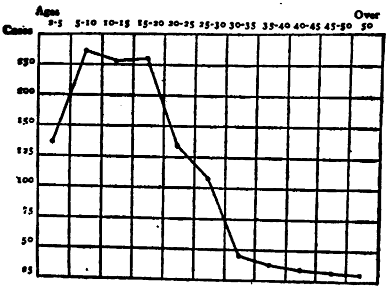
(1) Muench. med. Woch., No. 48, 1906, and No. 22, 1907.
(2) The Practitioner, April, 1907.

is bedridden, with contracted limbs." At the end the picture is exactly the counterpart of the acquired form in the adult, death occurring from bedsores or from some intercurrent ailment.

IDIOCY.

The general impression that idiots die young is not in accord with the facts, say L. Pierce Clark and C. E. Atwood.[1] This impression doubtless arises from the practice of showing only children in neurological clinics when idiocy is up for discussion. "Most text-books on nervous diseases make the general statement that idiots and imbeciles are short-lived. No official inquiry has been made into the subject, however, in idiot asylums or by bureaus of vital statistics; and, in fact, there is no literature on the subject. While it is true that life is markedly curtailed in the feeble-minded and idiotic, a very considerable number live beyond even the age of 40, as shown in the graphic chart of ages at death of the last thousand cases dying at the New York City Idiot Asylum, and a second chart illustrating the ages of the 785 patients at the Randall's Island hospitals and schools."



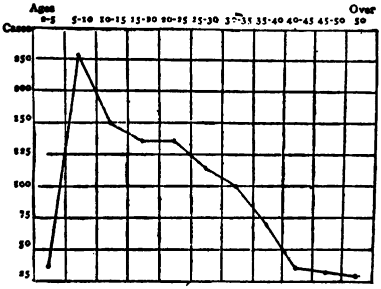


Chart 1 shows that while the maximum number of idiots died under the age of 25 years, thirty cases lived to be over 35, seventeen over 40, and four over 50 years of age. Of the twenty-one congenital idiots over 40 years of age now on Randall's Island, the photographs of four appear in Plates XV and XVI. "From the photographs, two interesting facts are at once apparent even to the nov-ice. First, all the subjects look much older than they really are; and, second, notwithstanding that we are told that idiocy implies an absence of facial expression, we note in these illustrative photographs a remarkably increased number of facial wrinkles."

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[1] Med. Rec., Aug. 31, 1907.

PARANOIA.

Paranoia, its varieties and medicolegal importance, is presented by Charles W. Burr.[1] "The paranoiac is the product not of environment, but of heredity. He is the victim of internal, inherent, congenital weakness. The inheritance is rarely direct; that is to say, a paranoiac parent rarely has paranoiac offspring, but always there is a bad strain in the family; some form of insanity, marked eccentricity, pseudogenius, epilepsy, hysteria, alcoholism, or criminality.

"The essential *symptoms* are supreme egotism, delusions of persecution and of grandeur which are always systematized and always reasoned about, absence of perversion of the moral sense, and an apparently strong but really weak intelligence. The important minor symptoms are hallucinations, falsification of memory and sexual perversions. A negative symptom of some importance is the absence of physical disease either of the central nervous system or of the abdominal or thoracic viscera. Anatomic abnormalities, the so-called physical stigmata of degeneration, are no more frequent than in the general population, though individual patients sometimes show many marked signs.

"More important than the mere list of symptoms is their mode of development and gradual evolution. The history of a case of paranoia is the biography of the man. As it is a disease of personality, the superficial aspect of any case varies from time to time, and, since the paranoiac has different moods, and since sometimes his delusions may submerge themselves, or he may consciously and voluntarily be able to hide them for a time, it is impossible to make a diagnosis from one examination unless the patient's previous history be known. To know a paranoiac, as some one has said, the physician must not

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(1) Jour. Am. Med. Assoc., June 1, 1907.

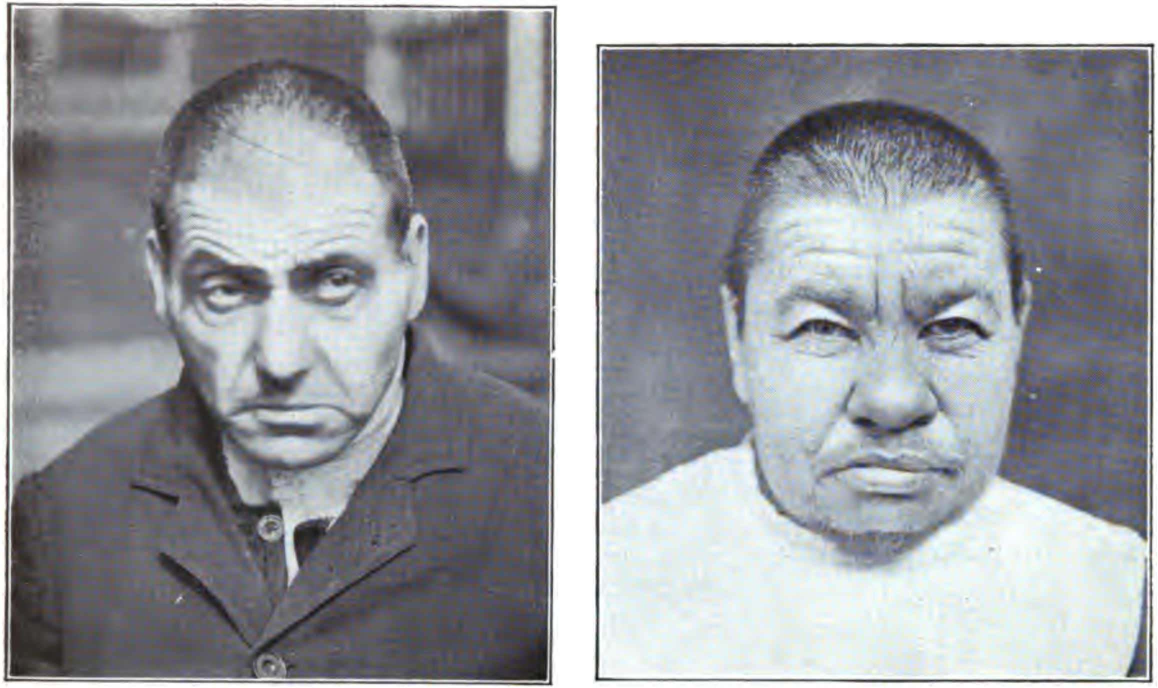

PLATE XVI.

A. Aaron S., aged 44 years.

B. John M., aged 40 years. (Page 223.)

make a study of a cross-section of his life (one day), but of a longitudinal section (many months, or even years). . . .

"Much is said of the intellectual strength of the paranoiac, but it is apparent rather than real. He is usually, but not always, in the earlier stages, a great talker, though some are sullen and silent throughout. He is always right, the rest of the world wrong. He is plausible and may make a great impression on those who know nothing of what he is talking about. He is cunning and for a purpose can hide his delusions for a time, but his vanity is his weakness, and by an appeal to it he can be induced to show what he thinks and believes.

"The moral sense lessens steadily from the beginning until it is absent altogether. The paranoiac has no feeling of altruism, and gets no pleasure from giving pleasure. Whether he becomes a criminal depends on circumstances and on whether or not he has the pseudocourage necessary to commit murder. He is always a liar, and frequently has perverted sexual instincts. Erotic and political paranoiacs are the type most prone to commit serious crime. . . .

"Paranoia never shortens life. Most patients live to old age; none recover. The intensity of the symptoms may vary from time to time, and the quiet and discipline of a well-regulated hospital may cause the delusions to become submerged temporarily, but they emerge again. Dementia finally ends the scene."

Albert W. Ferris[1] describes the manifestations of paranoia, naming its decisive characteristics as follows: (1) "The unchangeableness of the fundamental fixed idea; (2) the firm belief of the patient in his delusions; (3) the apparent (though unreal) logic of the delusional system; (4) the suddenness and intensity of the reactions; (5) the frequency of false interpretations; (6) the preservation for years of an intellect comparatively unimpaired, though dominated by delusion.

"Besides these essentials, there are in most cases interesting additional features, such as *expansive delusions,* the patient believing he is possessed of special charms, unusual strength, and remarkable influence, attributes which constitute the reason for the envy, malice, and persecution by others that exist in his imagination. Closely connected with these ideas is the feeling of *self-importance* on which is easily grafted a change of personality, producing the familiar reincarnation of an Old Testament notable, of a former sovereign, or of a military celebrity, etc.

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(1) Medical Record, Aug. 17, 1907.

"Very characteristic, too, are the hallucinations, which are generally a prominent feature at some time during the development of the psychosis. The most frequent variety is that of hearing, and it is very vivid and insistent. Most patients state that single words or short sentences are uttered in their hearing by strangers, servants, or unseen persons, and that the utterances are abusive, and directed toward themselves personally. Usually these accusatory remarks indicate to the patient that his whole history, as well as his innermost thoughts, are publicly known. Hallucinations of vision, smell, and touch are less frequent.

"The early manifestations of paranoia include irritability with excitement, studied disobedience, outward hostility, moodiness, desire for seclusion, perverted sexual instincts, dreamy indolence, a belief by the patient that he is misunderstood or slighted or not treated with as much consideration as the other children of the family, inordinate satisfaction with his own opinion, and a habit of demonstrating others' inferiority; periods of depression, possibly precocity. Such a syndrome of symtoms occurring in a youth should arouse the parents or tutor to a feeling of alarm, and open their eyes to the fact that, if not already present, there is a possibility of the development of mental disorder. He should be protected from the fatigue incident to long school hours, study at night, competitive examinations, children's parties, and exhausting games, as well as from improper food and indigestion.

"The reports of recovery from paranoia must be viewed with extreme suspicion. The disciplinary routine and the regularity of life as to eating, sleeping, exercise, and recreation incidental to custodial care in a proper hospital, exercise a very beneficial effect on the disease, and one of great value in modifying it. But the affection is incurable. The few cases that are reported as cured, and that have even been legally rehabilitated as sane people because of the absence of former delusions, should be under constant observation until their delusions return or dementia supervenes."

DEMENTIA PRECOX.

Adolf Meyer,[1] in an article on the fundamental conceptions of dementia precox, throws a good deal of light upon the subject, besides showing marked common sense in its consideration. He believes that there are very few who do not consider Kraepelin's creation of dementia precox the great psychiatric advance in recent times. It is time to think of creating disease entities among mental maladies, with due utilization of our knowledge of cause, course, and outcome. In the older text-books there is no truly helpful and convincing description of the factors which determine an unfavorable course in mental disorders. "Instead of the usual hazy statements, Kraepelin has pointed out features which are common to a great number of the so-called terminal dements, and he has found them to exist from the very beginning in a very large number of all cases that ultimately deteriorate. The emphasis of these features, somewhat at the expense of the temporary pictures which have so far received the main attention, was a real stroke of genius."

Kraepelin's bold picture allows of a correct estimate of a very large number of cases which formerly figured as doubtful with regard both to classification and outcome. Every alienist knows that he meets with cases in which from the beginning the prognosis is poor, and to a very large extent these cases are the ones stamped by the signs of Kraepelin's deterioration process. These signs are the appearances of negativism, or mannerisms, and of stereotypes, and especially of "Willensstörung." Adolf Meyer would describe as the essential features, disruption of judgment, without any evidence of intoxication or other delirium, or without the manic-depressive thinking disorder, or the foundation of the hysterical or epileptic disorder; discrepancies between the mood and the general reaction; peculiar attention; and an interference with

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(1) British Med. Jour., Sept. 29, 1906.

thinking and deterioration in matters largely dependent upon sound instinct, such as the differentiation of the real and the unreal—all these things in the face of a relative clearness, so that we are forced to think of a fundamental deterioration or defect as the only means to account for so much perversion of instinct and reasoning. In connection with this there appear a number of symptom-pictures, such as states of puzzle, of religious and mystic fascination, of automatic and stuporous states, such as can be in part obtained by hypnotic suggestion, or such as arise in psychasthenia and hysteria. The insufficiency of the provoking factor, and the oddity and incongruity rather than the mere excess constitute one of the most important criteria for the estimation of the seriousness of the process.

Every individual is capable of reacting to a very great variety of situations by a limited number of reaction types. "The full, wholesome, and complete reaction in any emergency or problem of activity is the final adjustment, complete or incomplete, but, at any rate, clearly planned, so as to give a feeling of satisfaction and completion. At other times there results merely an act of perplexity or an evasive substitution. Some of the reactions to emergencies or difficult situations are mere temporizing, attempts to tide over the difficulty, based on the hope that new interests may crowd out what would be fruitless worry or disappointment. Complete or incomplete forgetting is the most usual remedy of the results of failures, and just as inattention and distraction correct a tendency to overwork, so fault-finding with others, or imaginative thoughts or praying, or other expedients, are relied upon to help over a disappointment, and, as a rule, successfully. Other responses are much more apt to become harmful, dangerous, uncontrollable—a rattled fumbling, or a tantrum, or a hysterical fit, or a merely partial suppression, an undercurrent, an uncorrected false lingering attitude, or whatever the reaction type of the individual may be.

"In the cases that tend to go to deterioration, certain types of reactions occur in such frequency as to constitute almost pathognomic empirical units. I would mention hypochondriacal trends, ideas of reference, fault-finding or suspicions, or attempts to get over things with empty harping, unaccountable dream-like frequently nocturnal episodes, often with fear and hallucinations, and leading to strange conduct, such as the running out into the street in nightdress, etc., or ideas of strange possessions with hallucinatory dissociations, or the occurrence of fantastic notions. All these appear either on the ground of a neurasthenoid development, or at times suddenly, on more or less insufficient provocation, with insufficient excuse, but often enough with evidence that the patient was habitually dreamy, dependent in his adjustment to the situations of the world rather on shirking than on an active aggressive management, scattered and distracted either in all the spheres of habits, or at least in some of the essential domains of adjustment which must depend more or less on instinct or habit. On this ground, reaction types, which also occur in milder forms of inadequacy, in psychasthenia and hysteria or in religious ecstacy, etc., turn up on more inadequate foundation and with destructive rather than helpful results. We thus obtain the negativism no longer as healthy indifference and more or less self-sparing dodging, but distinctly as an uncontrollable, unreasoning blocking factor. We obtain stereotypes not merely as substitutive reactions and automatisms on sufficient cause such as everybody will have, but, as it were, as a reaction of dead principle in a rut of least resistance. We see paranoiac developments with the same inadequacy of starting point and failure in systematization, and in holding together the shattered personality, etc.

"There is no doubt that many diagnoses of dementia precox are made on the ground of rule-of-thumb principles. This danger can be obviated by our taking a definite view of what diagnosis in mental disease means. Where it is on safe and easily controlled ground, the diagnosis is plainly a brief term for the statement of fact. As one gets into ill-defined facts, the diagnosis becomes an expression for a substitutive attitude rather than for the actually demonstrated facts. A diagnosis usually does justice only to one part of the facts, and is merely a convenience of nomenclature. In psychiatry, the facts occur in very complex combinations, and therefore a one-word diagnosis is almost sure to fall short of presenting the actual facts in the case sufficiently to designate the etiological, symptomatic, prognostic, and therapeutic status. Psychiatry has not reached, and probably never will reach, the stage where a small number of one-word diagnoses would be more than a formal index.

"We should bear in mind that the concept of dementia precox had better be looked upon as designating a perspective of deterioration. In many cases the deterioration is, however, not imminent, nor the most interesting feature; it is a result to be borne in mind and dreaded, but not as directly interesting as the process which threatens miscarriage. In a fairly large number of cases we should, therefore, designate the process not with the term of a possible or even probable outcome, but with one designating the principle at work—the hysteroid reaction, the abnormal habit reaction, the type of attention defect and of judgment defect. The subforms, such as the hebephrenic and the katatonic and the paranoid, will in the main preserve a standing, but they will appear less important as we shall learn to single out the determining factors. As our knowledge will increase, combination, terms designating etiology and reaction-type, and possibly a measure of the balance, will take the place of the as yet rather schematic types; but for the time being the small number of types taken over by Kraepelin from Kahlbaum and Hecker will be used without much hair-splitting.

"*Therapeutically,* this way of going at the cases will furnish the best possible perspectives for action. We stand here at the beginning of a change which will make psychiatry interesting to the family physician and practitioner. As long as consumption was the leading concept of the dreaded condition of tuberculosis, its recognition often came too late to make therapeutics tell. If dementia is the leading concept of a disorder, its recognition is the declaration of bankruptcy. Today the physician thinks in terms of tuberculous infection, in terms of what favors its development or suppression; and long before "consumption" comes to one's mind, the right principle of action is at hand. In the same way, a knowledge of the working factors of dementia precox will put us into a position of action, of habit-training, and of regulation of mental and physical hygiene, as long as the possible 'mental consumption' is merely a perspective and not an accomplished fact."

Smith Ely Jelliffe[1] discusses the signs of what he calls *predementia precox,* their significance, and pedagogic prophylaxis. In a general way he believes that it is possible, by taking heed of little things, to conserve in some degree, sufficient for ordinary needs, an individual who otherwise would pass to utter destruction. More attention should be given by physicians to puberty. This is the period of sudden expansion of the personality and individuality. Self-consciousness appears, reflectiveness may lead to morbid self-criticism; often furtiveness and oversuspiciousness present themselves. With others, castle-building is the chief occupation, and there are thoughts of overesteem, overconfidence in one's ability, which may lead to insane conceits. Imagination may be boundless. Illusions are many and varied, and often their making becomes so pleasure-giving a device that it cannot be thwarted.

"Inasmuch as youth has not gathered enough facts of life on which to base wide judgments, it is very natural that at this period, when some reaction is necessary, we should find a sudden blossoming out of the imitative faculty. This in the motor sphere shows itself very distinctly in mannerisms, modes of walking, etc. In a small number one sees these reactions become stereotyped—as it has been well expressed, just as speech may lose its highest function and lapse to not only slang, but even interjectional forms, so poses may slowly become fixed postures and even contractures, or a limited repertory of stereotyped acts may develop on a basis of the natural automatisms of Lindley. . . .

"Looking back over a series of years and reviewing the early histories of a comparatively large number of patients whom I have known fairly intimately, my own observation has led me to note that most of the precocious dements with the paranoid tinge have occurred in those whom I have known to be singularly lacking in the divertibility of the attention, particularly for objects of auditory and optic sense. These young people I have known have all been very poorly gifted in the power of external ob-

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(1) Amer. Jour. of the Med. Sciences, Aug., 1907.

servation. Their lights were all turned within, and while all were singularly gifted, their gifts were made manifest in matters of literary or artistic constructive qualities, using the products of introspection rather than in the direction of their being natural science workers. This observation is in need of careful scrutiny, and is of the broadest character and is recorded simply as such."

Heredity is important in dementia precox, which among all the psychoses shows the largest number of abnormal antecedents, even exceeding the proportions of manic-depressive insanity. Kraepelin places the percentage of those showing antecedents definitely tainted at 70, and in at least 17 to 18 per cent. the immediate ancestry, paternal or maternal, is involved. Krauss, in 1903, working with the material of the Heidelberg Clinic, states that he has found as high as 93 per cent. of hereditary taint in dementia precox among both ascendants and descendants, but his figures are based upon a small number of cases.

Jelliffe's own observations have led him to the conclusion that three elements are most emphatic in the ancestry: dementia precox itself, alcohol, and abnormal personality, or crankiness, if it may be so called. In his experience, alcoholic parents have been responsible chiefly for hebephrenics. Marital incompatibility was a significant factor in many of the panarnoid-dements in patients known to Jelliffe in the pre-dementia stages. Another class of antecedents may be termed the "derelicts,"—themselves in all probability suffering in some slight degree from dementia precox coming on in later life. Many of these are the semi-failures of life,—those pushed aside in the struggle. This class has contributed the largest number of all classes of precocious dements to Jelliffe's cases.

"As to the influence of inbreeding, experience has shown that it all depends on those that breed. If neuropathic trends are present in those of blood relationship, it is much more likely than not that the children will suffer. Healthy stock intermarrying often shows excellent progeny. . . . Inbreeding is bad only when the characters bred are in themselves distinctly pathological."

An additional factor besides heredity in the production of dementia precox is tire, or fatigue, concerning which there is a diversity of opinion. In general, the natural adjustment of the normal adolescent to the influence of fatigue (laziness, distraction, etc.) will prove sufficiently protective, yet for the adolescent with hereditary taint, fatigue is a highly important element in the mental breakdown. This fact is well supported by the psychological studies upon the influence of fatigue. The symptoms developing in children with tainted heredity as a result of overwork plus other indeterminate factors are very striking.

Prophylaxis. The point of view adopted by Jelliffe leads to the assumption that enlightened teaching can prevent mental breakdown. It is folly to believe that mental deterioration may be entirely avoided by proper teaching, yet the very protest of those who object to the term dementia precox because it implies a hopeless prognosis, whereas many recover, indicates quite clearly that a slight degree of abnormal mental functioning, which might, if left to itself, subsequently progress into frank dementia precox, can be radically improved by early recognition and the institution of proper physiological and pedagogic correctives. It is self-evident that children of abnormal parents should be brought up with more than the usual amount of care, particularly during puberty and early adolescence. Often such children are nervous, many showing disturbances in sleep, such as restlessness in sleep, night-terrors, or even convulsive seizures accompanied by cries and apparent hallucinatory influences. Other signs of nervousness are not to be overlooked, such as attacks of sudden anger or passion, coming out of a clear sky, excessive reactions to emotional causes, continual dissatisfaction, imperative ideas and imperative actions, impulsive desires to steal, to hoard trivial things. If to these things are added attempts to set fire to buildings, strong sexual excitement leading the exhibitionism, a proclivity to continual running away from home, the problem is a serious one.

How shall these resisting, self-willed, often cruel or tantalizing young children be corrected? Whipping is undeniably bad. Any brutal method of punishment drives them more and more within themselves.' Suspiciousness crops out, and may lay the foundation for a paranoid coloring in later life. Normal natural resentment leads to sudden and violent outbursts, almost maniacal in violence.

"Probably the most important element in the prophylaxis of dementia precox for the nation is the forbidding of marriage. The children of such patients almost invariably bear a taint. Nevertheless, the physician seldom has the opportunity to exert his influence, for parents are not in the habit of consulting a specialist to ask whether a hysterical, excitable daughter ought to marry, or whether the ungovernable temper of a son means inequilibrium."

Children showing signs of pre-dementia precox should lead a simple outdoor life. "My own observation confirms my belief that this method, as usually practiced, is distinctly harmful, for the usual idea underlying its application is that the air will do everything." Air is a comparative negligible quantity. Proper care and surroundings are the main factors, and in prescribing country air these are never to be lost sight of. Sports are highly desirable, but not competitive sports. Rowing, skating, swimming and horseback riding are to be classed in the front rank. Education by industrial occupation is especially useful. "Competitive intellectual pursuits are to be carefully avoided. The working out of complex themes, steady composition, much arithmetical calculation, are undesirable. Debating, memorizing, and dramatics, I consider harmful." For a life pursuit, agriculture offers the best opportunity, and Jelliffe states that he has personally known of "a number of men, who, recognizing their bad mental heritage and perceiving while still young the strain of urban competition, have changed their mode of life entirely, have taken up agricultural pursuits and are now after twenty years well, strong, and happy, and some even have acquired more than a modest competence."

INDEX.

Abscess, cerebral, 94.
  diagnosis, 96.
  etiology, 96.
  symptomatology, 94, 95.
    hippus in, 94.
Acroparesthesia, 196.
  nocturna, 198.
  treatment, 197.
Adiposis dolorosa, 201.
Amaurotic family idiocy, 207.
  allied cases, 208.
Amnesia, tactile, 68.
  cortical seat, 69.
Amyotrophic lateral sclerosis, 154.
Aphasia, 73.
  intellectual deficit in, 78.
  localization, 76, 77.
Apoplexy, 84.
Arteries, syphilis of, 89.
Arteriosclerosis, 79, 138.
  cerebral, 79.
  spinal, 138.
Astereognosis, 69.
Ataxia, auditory, 8.
  cerebellar, 122.
  Friedreich's hereditary, 154.
Atrophy, progressive muscular, 202.
Auditory nerve, disease of, 176.

Bell's palsy, 174.
Beriberi, 193.
Birth palsy,
  brachial, 185, 187.
    surgical treatment, 185, 188.
    symptoms, 187.
  cerebral, 83.
Brachial birth palsy, 185.
Brachial neuralgia, 180.
  treatment, 184.
Brachial plexus, paralysis, 187.
Cerebellum, diseases of, 120, 125.
  functions of, 120.
  hemorrhage into, 125.
  peduncle, superior, destruction of, 127.
  symptoms of disease of, 122, 126.
  tumor of, 128.
    cerebello-pontine, 131.
    middle lobe, 120.
    reflexes in, 129.
    symptoms of, 128, 130.
    treatment of, 133.
Cerebrospinal meningitis, 41.
  (See also meningitis, cerebrospinal.)
  cytodiagnosis in, 9.
  diplococcic, 41.
  rheumatic, 61.
  streptococcic, 62.
  tuberculous, 63.
  treatment of, 54-59.
Chorea, hereditary, 200.
  Huntington's, 199.
  Sydenham's, 37.
    insaniens, 40.
    symptoms, 37.
    treatment, 41.
Conjugate deviation of eyes and head, 70.
Cord, syphilis of, 159.
Corpus callosum, tumors, 113.
Cortex, frontal lobes, 65.
  localization of, 64.
  motor, 66, 67.
  parietal lobes, 68.
  prefrontal lobes, 64.
Cranial nerves, 169.
Cytodiagnosis, 9.

Decompression, cerebral, 115.
Dercum's Disease, 201.

Dementia precox, 227.
  early stages, 231.
Diplococcus intracellularis, 41.
  agglutination and opsonic reaction, 41, 45, 47, 59.
  in the blood, 49.

Embolism, cerebral, 88.
  treatment, 89.
Encephalitis, 93.
Endarteritis syphilitica, 89.
Epilepsy, 30.

Facial paralysis, 174.
Facial spasm, 176.
Friedreich's hereditary ataxia, 154.
  differential diagnosis, 155.
  pathology, 156.
Fugues, hysterical, 24.

General paralysis, 219.
  classification, 219.
  in old age, 219.
  juvenile form, 222.
  symptomatology, 219.
  treatment, 221.
Giantism, 201.

Hemiplegia, 84.
  convulsions in, 86.
  differential diagnosis, 84.
  involvement of jaw muscles, 92.
  in uremia, 91.
Hemorrhage, cerebral, 85.
  diagnosis and treatment, 85.
Herpes, auricularis, 175.
  ophthalmicus, 169.
Huntington's chorea, 199.
Hydrocephalus internus, 98.
Hypoglossal nerve injury, 178.
Hypophysis cerebri, disease of, 201.
  tumor of, 114.
Hysteria, 23.
  fugues in, 24.
  in children, 23.
  joints in, 25.
  paralytic ptosis in, 25.
  treatment, 28.

Idiocy,
  amaurotic family, 207.
  in old age, 223.
Indormescent shocks, 198.
Ingravescence in hemiplegia, 87.
Insanity, 210.
  general prognosis, 212.
  increase of, 210.
  occupation therapy, 218.
  prophylaxis, 213, 214.
  treatment, 216.

Joints, hysterical, 25.
  syringomyelic, 163.
  tabetic, 139.
Juvenile general paralysis, 222.

Landry's paralysis, 148.
  bacteria, 149, 152.
  differential diagnosis, 149, 150.
  pathology, 148, 153.
  recovery in, 152.
  symptomatology, 153.
Language zone, location, 77.
Light reflex, secondary, 220.

Medulla, trauma of, 134.
Meningitis, cerebrospinal, epidemic, 41.
  epidemics of, 50, 51.
  mode of infection, 42.
  symptomatology, 50, 53.
  treatment, 53.
Meningitis, posterior basic, 54.
Meningitis, purulent,
  cytodiagnosis in, 12.
Meningitis, tuberculous,
  cerebrospinal fluid in, 11.
Migraine, 35.
Miscellaneous nervous diseases, 196.
Multiple neuritis, 191.
Multiple sclerosis, 156.
  differential diagnosis from syphilis, 159.
  pathology, 156.
  prognosis, 160.
  symptomatology, 157.
Myatonia congenita, 203.
Myelitis, acute ascending, 141.
  transverse, 136.
    indications for operation, 137.
    symptoms, 136.

Nerves, degeneration and regeneration, 5.
  cranial, 169.
  spinal, 179.
Neuralgia, brachial, 180.
  trigeminal, 170.
    treatment, 170, 172, 173.
Neurasthenia, cerebellar, 25.
  general, 19.
    treatment, 22.
  tropical, 21.
Neuritis, pathogenesis, 179.
Neurone theory, 5.

Optic thalamus, syndrome, 119.
  tumors of, 112.

Painful awakenings, 198.
Pallesthesia, 6.
Paralysis agitans, 205.
  without tremor, 206.
Paranoia, 224.
  early signs, 226.
Peroneal nerve paralysis, 190.
Pneumogastric nerve, disease of, 177.
Poliomyelitis acuta, 143.
  bacteria in, 145.
  relapsing form, 145.
  symptoms, 143, 146.
  treatment, 146.
Polyneuritis, see multiple neuritis.
Polyneuritis cerebralis acuta, 176.
Predementia precox, 231.
  prophylaxis, 233.
Progressive muscular atrophy, 202.
Ptosis, in hysteria, 25.

Quinquaud's sign, 7.

Raynaud's disease, 199.
Reflexes, 13, 15.
  cerebral element in, 13.
  fatigue in, 17.
  in cerebellar tumors, 129.
  inhibition, 16.
  paradoxic of Gordon, 15.

Sarcoma, primary melanotic of spinal cord, 167.
Sciatica, treatment, 190.
Senility, 80.
Sinus thrombosis, 93.
Sleep, disorders of, 198.
Spasm, facial, 176.
Spinal cord,
  pathway of sensory impulses, 135.
  trauma, 136.
  tumors, 164.
    primary melanotic sarcoma, 167.
    symptoms, 164.
    treatment, 165.
    tuberculoma, 166.
Spinal nerves, 179.
Syphilis, of arteries, 89.
  of cord, 159.
Syringomyelia, 160.
  arthropathy, 163.
  differential diagnosis, 163.
  dissociation symptom, 135.
  pathology, 160, 161.
  symptomatology, 162.

Tabes dorsalis, 138.
  arthropathies, 139.
  etiology, 138.
  pregnancy in, 140.
  symptomatology, 138.
  treatment, 140.
Temporal lobe, tumors, 110.
Thrombosis, cerebral, 87.
Transverse myelitis, traumatic, treatment of vesical paralysis, 192.
Trigeminal nerve, diseases of, 169.
Trophedema, 202.
Tuberculoma of spinal cord, 166.
Tumors,
  cerebellar, 128.
    cerebello-pontine angle, 131.
  cerebral, 98.
    diagnosis, 98.
    enchondroma, 101.
      multiple, 102.
    epithelioma, 102.
    localization of, 103, 107.
    of corpus callosum, 113.
    of frontal lobe, 103, 105, 107.

    of optic thalamus, 112.
    of supramarginal and angular gyri, 110.
    of temporal lobe, 110.
    treatment, 114.
      by decompression, 115.
  spinal cord, 164.
    diagnosis, 168.
    symptoms, absence of pain, 168.
    treatment, 168.

Typhlolexia congenita, 79.

Uremia, hemiplegia in, 91.

Vagal attacks, 177.
Vasovagal attacks, 177.
  treatment, 178.

X-ray in diagnosis of hysterical joints, 27.
  in treatment of syringomyelia, 164.

INDEX OF AUTHORS.

Allan, G. A., 53.
Albutt, C., 54.
Allen, A. R., 98.
Allison, N., 146.
Alquier, L., 163.
Amblard, L. A., 79.
Atwood, C. E., 83, 102, 146, 202, 223.

Babes, V., 221.
Babinski, J., 41, 135.
Bard, 71, 72.
Barker, L. F., 25, 139, 163.
Barlow T., 54.
Barnes, S., 145.
Batten, F. E., 164.
Baudouin, 170, 172.
Beaujard, E., 164.
Beevor, C. E., 107, 110, 112, 128.
Bennion, J. M., 32.
Bernhardt, M., 140, 205.
Bernheim, F., 73.
Bernstein, J., 152.
Bethe, 6.
Borchardt, M., 131.
Bourret, C., 94.
Bowers, W. G., 140.
Brissaud, 172, 190.
Brown, S., 157.
Burr, C. W., 68, 224.
Buzzard, E. F., 141, 143, 148.

Camp, C. D., 159, 205.
Camus, P., 139.
Cestan, R., 93, 102.
Charpentier, A., 40.
Clark, L. P., 102, 146, 174, 202, 223.
Claude, H., 25, 30.
Cleghorn, C. D., 19.
Collins, J., 79, 138, 196.
Comrie, J. D., 42.
Cook, R. G., 95.
Coombs, C., 203.
Cushing, H., 83, 114.

Da Costa, 15.
Dana, C. L., 76, 96, 122, 180.
Déjerine, J., 119, 156, 174.
Dercum, F. X., 15, 73.
Devic, 134.
Diller, T., 131.
Douglas, J. S. C., 63.
Ducoste, M., 119.
Dudgeon, L. S., 59, 145.
Dunn, J. S., 53.

Eshner, A. A., 206.
Etienne, G., 202.

Fales, L. H., 21, 193.
Fennell, C. H., 222.
Ferris, A. W., 225.
Filipkievitz, S., 138.
Finny, J. M., 40.
Flexner, S., 57.
Florian, S., 202.
Förster, O., 38.
Fränkel, 74.
Franz, S. J., 65.
Fraser, J. S., 42.
Frazier, C. H., 115, 116.

Gay, W., 41.
Gendron, A., 92.
Gittings, J. C., 203.

Gordinier, H. C., 66.
Gordon, A., 15, 22, 31, 64, 68, 166, 208.
Gowers, Sir W., 35, 85, 108, 177.
Griffith, T. W., 154.
Grinker, J., 38, 191.
Gwyn, 49.
Gy, A., 25.

Hall, J. N., 153.
Halliburton, W. D., 5.
Hamilton, A. S., 199.
Haskovec, L., 201.
Hauck, L., 172, 190.
Hay, A. G., 190.
Head, 135.
Hecht, D., 23.
Heitz, J., 140.
Hoffmann, 221.
Holmes, G., 207.
Hopkins, S. D., 153.
Hoppe, H. H., 99.
Horsley, V., 115, 120.
Houston, T., 45.
Hudovernig, C., 201.
Hunt, J. R., 175.

Jackson, J. H., 110, 120.
Jacob, L., 152.
Jacoby, G. W., 184.
Jastrowitch, 65.
Jehle, 42.
Jelliffe, S. E., 179, 231.

Kilvington, B., 192.
Kindler, E., 141.
King, C., 200.
Kirby, G. H., 216, 220.
Koplik, H., 55.
Kraepelin, 216, 227, 232.
Kutscher, H. H., 47.

Langmead, F., 54.
Laqueur, A., 140.
Lecomte, 79.
Lépine, 61.

Léri, M. A., 80.
Lesem, W. W., 196.
Leszynsky, W., 99, 114.
Lévy, F., 170, 172, 176.
Lewandowsky, M., 152.
Lhermite, J., 163, 164.
Lloyd, J. H., 67, 190.
Loedlein, M., 96.
Long, E., 135, 202.

McEwen, 56.
McLaren, H., 161.
Maas, O., 160.
Macdonald, J. A., 101.
Mackenzie, H., 58.
Macnamara, A., 152.
Mann, 153.
Marie, P., 64, 73.
Mason, J. M., 51.
Meyer, A., 227.
Meyers, C., 213.
Miller, J., 145.
Miller, R., 145.
Mills, C. K., 72.
Minor, L., 7.
Mirallié, C., 92.
Mober, T. J., 218.
Monro, T. K., 161.
Moschcowitz, A. V., 173.
Mottram, J. C., 59.
Müller, E., 158.
Murat, L., 93.

Nambu, T., 156.
Nedwill, C. L., 50.
New, F. C., 101.
Noeggerath, C. F., 63.
Nordmann, 134.

Olivier, M., 219.
Onuff, 74.
Oppenheim, 65, 130, 160, 203
Orr, D., 179.
Osler, W., 49, 200.

Parhon, G., 202.
Parsons, J. H., 169, 207.
Patrick, H. T., 24, 34, 170, 172, 198.
Paul, W. E., 13.
Perkins, J. J., 145.
Perthes, 173.
Phelps, 104, 200.
Porot, A., 127.
Potts, B. H., 96.
Poynton, F. J., 207.
Price, G. E., 39.
Prout, T. P., 185, 187.
Prunier, A., 201.
Putnam, J. J., 113.

Rankin, J. C., 45.
Raymond, 93, 102, 113, 164.
Rhein, J. H. W., 91.
Riebold, G., 63.
Riggs, C. E., 94.
Robertson, W., 42.
Roscher, 221.
Rose, 25.
Rose, L. W., 95.
Ross, N. B., 30.
Rous, F. P., 9.
Rows, R. G., 179.
Rundle, C., 59.

Sachs, B., 130, 133, 208.
Salomon, 49.
Sarvonat, F., 199.
Sauvineau, C., 25.
Savage, G. H., 210.
Savill, T. D., 28.
Schlesinger, 102, 161.
Schlösser, 170, 173, 176, 190.
Schmaus, 153.
Schoenborn, S., 176.
Schultze, F., 168.
Sérieux, P., 219.
Sézary, A., 139.
Shanahan, W. T., 130.
Sherrington, C. S., 15, 73.
Shimer, I. A., 194.
Sicard, 172, 190.
Simon, C. E., 49.
Sinkler, W., 152.
Smith, W. G., 15.
Southard, E. E., 62.
Spielmeyer, W., 138, 209, 221, 222.
Spiller, W. G., 73, 98, 115, 159, 160, 203, 209.
Stadelmann, E., 152.
Starr, M. A., 33.
Stewart, T. G., 103.
Stiles, F. M., 30.
Still, 54.
Stratton, R. R., 62.

Tanon, 172, 190.
Tay, W., 208.
Taylor, A., 47.
Taylor, A. S., 185, 187.
Thayer, W. S., 37.
Thomas, A., 156.
Thompson, 135.
Thompson, H. C., 158.
Thomson, A., 136.
Thorel, C., 167.
Timme, W., 100.
Tobias, E., 141.
Todd, 38.
Toogood, F. S., 214.

Uhlenhut, 221.
Urquhart, A. R., 212.

Variot, 79.
Vaschide, N., 78.
Vasilin, A., 221.

Walton, G. L., 13, 98, 219.
Warrington, W. B., 156, 187.
Weisenburg, T. H., 32, 70, 119.
Wheat, J. M., 195.
Wilkes, W. O., 54.
Williams, R. S., 59.
Williamson, R. T., 6, 154.
Wimmer, A., 93.
Wright, A. E., 48.
Wright, W., 54.

Zacharias, P., 140.